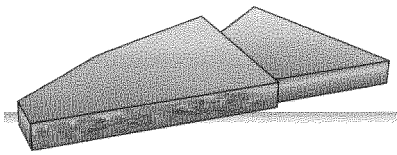

6 BASELINE RISK ASSESSMENT

The ABRA evaluated potential risks to human health and the environment from contaminants buried at the SDA. The risk assessment approach was based on EPA and INEEL guidance (EPA 1989; LMITCO 1995c). A comprehensive approach was used to evaluate WAG 7 risk. That is, cumulative health effects were assessed for all complete pathways for the entire SDA. This risk assessment builds on the work presented in the IRA (Becker et al. 1998). A source term model (see Section 5.1) was used to estimate contaminant releases into the environment for the COPCs identified in Section 3.4. Additional long-lived radioactive decay products were assessed for completeness. For groundwater pathway analysis, a three-dimensional model was used to estimate potential groundwater concentrations (see Section 5.2). Volatile organic compound risks were developed by scaling the concentrations and risk estimates from the IRA, as explained in detail in Section 5.3. For the human health soil exposure pathways and the ecological risk assessment, the concentrations derived from modeling biotic intrusion into the waste were used to assess the cumulative health effects (see Section 5.5). All the modeling is discussed in Section 5.

The majority of Section 6 is directed specifically at assessing human health risks in Sections 6.1 through 6.5. The ecological risk assessment is presented in Section 6.6. The components of the risk analysis are described under the general headings listed below:

- Section 6.1—Assumptions for the baseline risk assessment
- Section 6.2—Human health exposure assessment
- Section 6.3—Toxicity profiles for human health COPCs
- Section 6.4—Human health risk characterization
- Section 6.5—Uncertainty analysis
- Section 6.6—Ecological risk analysis
- Section 6.7—References cited in this section.

6.1 Assumptions for Baseline Risk Assessment

Assumptions specific to developing human health risk estimates are discussed below. Assumptions related to source release, fate and transport, and biotic modeling are discussed in Section 5. The DOE land use projections incorporate an assumption that institutional control will be maintained at the INEEL for at least 100 years (DOE-ID 1996). The same assumption was adopted as a basis for the ABRA. For this assessment, the 100-year simulated institutional control period was assumed to begin in 2010. Occupational exposure was evaluated for a total of 158 years to encompass SDA operations beginning in 1952 and ending in 2110, at the end of the simulated 100-year institutional control period. Current monitoring precludes the drinking of contaminated groundwater during the occupational scenario. Therefore, occupational exposures are limited to soil ingestion, dermal contact with soil, inhalation of particulates and vapors, and exposure to ionizing radiation.

An additional 900 years of residential exposure was simulated for all pathways, which include inhalation of particulates and vapors, soil ingestion, groundwater ingestion, ingestion of homegrown produce, dermal contact with organic chemicals both from the soil and while showering, and direct exposure to ionizing radiation. The following assumptions were incorporated into the ABRA.

- Residential receptors will be located at the nearest downgradient edge of the INEEL during the simulated 100-year institutional control period from 2010 to 2110.
- Residential receptors will be located on the SDA but will not intrude directly into the waste after the 100-year simulated institutional control period. This is equivalent to having the receptor living at the edge of the SDA. The receptor will be exposed to the average SDA soil concentrations and maximum groundwater concentrations. Intrusion was assessed qualitatively.
- Occupational receptors are located on the SDA.
- Nonradioactive contaminants do not degrade. The only mechanisms that reduce risk over time are radioactive decay and COPC concentrations diminishing through transport.

All pathways were simulated for 1,000 years from closure (2010). Because groundwater ingestion risk might not peak in that 1,000-year simulation period, groundwater is simulated for 10,000 years. Results are shown for 1,000 years for all pathways and 10,000 years for groundwater ingestion.

6.2 Human Health Exposure Assessment

In the human health exposure assessment, receptor intake of COPCs was quantified for all complete exposure pathways. The assessment consisted of estimating the magnitudes, frequencies, durations, and exposure routes of COPCs to humans. The following activities were performed as part of the exposure assessment:

- Identifying and characterizing exposed populations
- Evaluating exposure pathways
- Estimating contaminant concentrations at points of exposure for soil, air, and groundwater
- Estimating contaminant intakes.

The first two tasks are discussed together in Section 6.2.1. Media concentrations are discussed in Section 6.2.2. The quantification of the exposure is performed in Section 6.2.3.

6.2.1 Exposure Scenarios and Conceptual Site Model

The current occupational exposure scenario is used for modeling the timeframe from the opening of the SDA in 1952, through SDA closure in 2010, and until the end of the simulated institutional control period in 2110. (See Section 6.1.) A small portion of the SDA, Pits 17 through 20, is currently used for LLW disposal. During the simulated 100-year institutional control period, the occupational scenario is the only realistic exposure scenario for WAG 7 because institutional controls will prevent public access to the SDA. Therefore, in the ABRA, an occupational scenario was assessed for 100 years after closure. For this analysis, closure is assumed to occur in 2010, a total of 58 years from the start of waste burial operations at the SDA. However, because contaminants could leach into groundwater and be transported off-Site, residential groundwater risk at the INEEL boundary also was assessed for the simulated 100-year

institutional control period. Other residential exposure scenarios are not applicable for the institutional control period and were not evaluated.

A residential scenario was evaluated for 900 years following the simulated 100-year institutional control period. All pathways were assessed. The 900 years of residential scenario combined with 100 years of simulated institutional control provide the 1,000-year simulations for the pathway analysis. However, many contaminants do not reach ***peak*** concentration in groundwater within this timeframe. Therefore, the groundwater simulations were continued to 10,000 years (or the peak) and are presented separately.

The human health conceptual site model in Figure 6-1 shows complete exposure pathways for residential scenarios. Groundwater, air, and soil pathways are complete for residential exposures. The conceptual site model also shows some of the complexities in the exposure scenarios. For example, contaminated groundwater is directly ingested and also is used to irrigate crops, shower, and cook.

The conceptual site model shows that the groundwater pathway is incomplete for the occupational scenario because current operational procedures preclude the use of contaminated water as a drinking source. Complete occupational scenario exposure pathways include inhalation, soil ingestion, and direct exposure to ionizing radiation.

The ABRA addressed potential impacts of waste buried in the SDA but did not address past operational or flooding releases to the surface. Because perched water is ephemeral at the SDA, it is not considered a viable drinking water source. Therefore, perched water is an incomplete pathway for the analysis.

The following human health exposure routes (see Figure 6-1) were evaluated:

- Soil ingestion
- Inhalation of fugitive dust
- Inhalation of volatiles (includes residential scenario indoor use of groundwater)
- External radiation exposure
- Dermal absorption from soil (organic contaminants only)
- Groundwater ingestion (residential scenario only)
- Ingestion of homegrown produce (residential scenario only)
- Dermal absorption of contaminants in groundwater (residential scenario only).

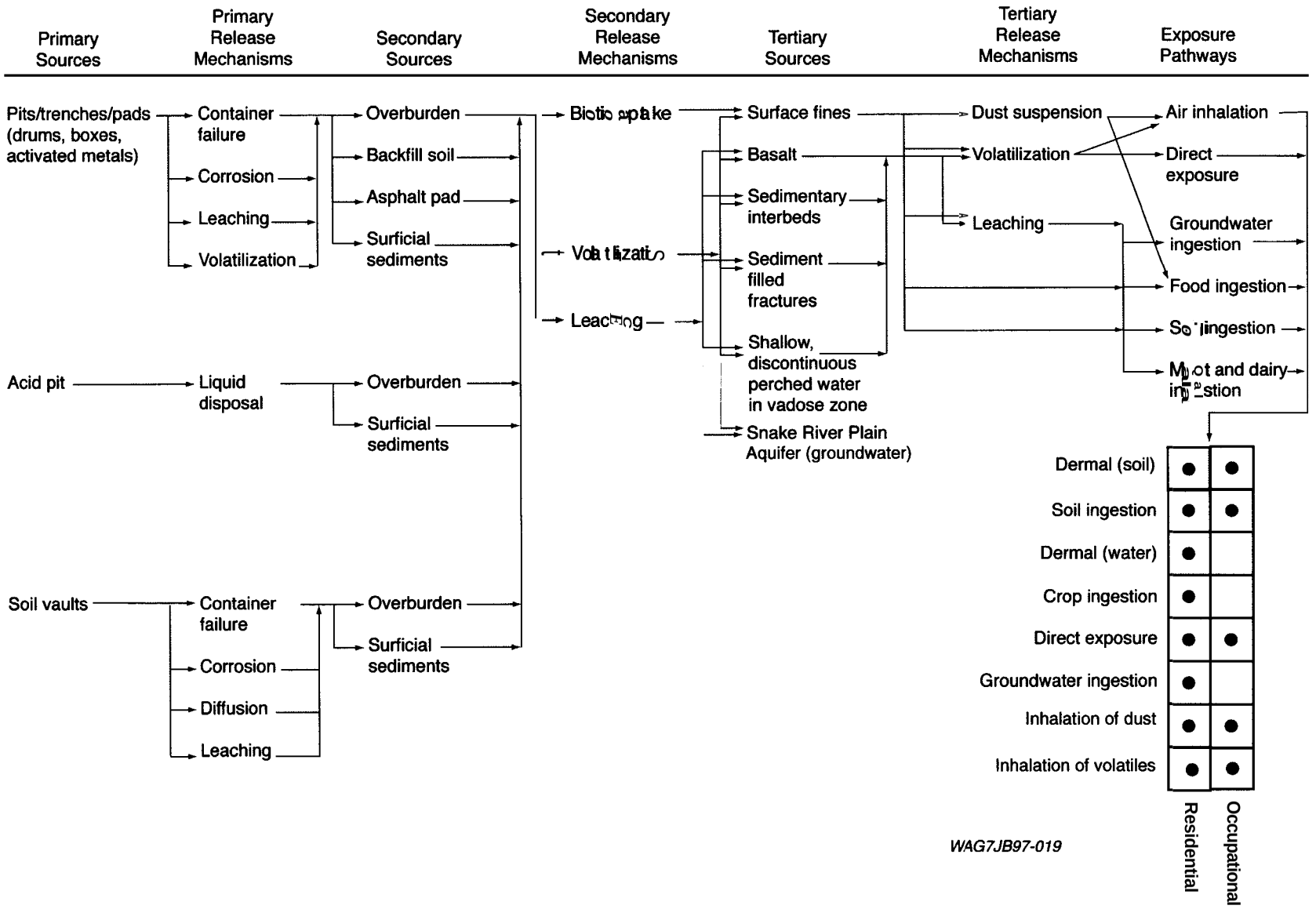

Figure 6-1. Human health conceptual site model.

6.2.2 Media Concentrations

Media concentrations were estimated by the modeling discussed in Section 5. The biotic transport code DOSTOMAN was used to estimate average concentrations of COPCs at the surface and at shallow depths down to *2.2* m *(7.2* ft) for the entire SDA. The source release code DUST-MS was used to simulate release of COPCs from buried waste into the subsurface beneath the SDA, and the resulting fluxes were input to the subsurface model TETRAD. The subsurface model simulated vadose zone transport and emulated COPC fluxes into the aquifer. TETRAD also was used to estimate concentrations and transport of COPCs in the aquifer.

Estimated media concentrations were used directly, (e.g., groundwater concentrations for the groundwater ingestion route) and indirectly (e.g., to develop media concentrations for other pathways such as air concentrations) to assess risk. The development of media concentrations for each exposure route is summarized in the sections below.

6.2.2.1 Soil Ingestion. Soil concentrations used to estimate the risk from ingesting contaminated soil were taken from the biotic model DOSTOMAN. The biotic model was used to predict surface soil concentrations in the SDA. Twenty-five-year average concentrations were used for the occupational exposure. Thirty-year averages were used for residential exposure.

6.2.2.2 Inhalation of Fugitive Dust. Soil concentrations produced by the DOSTOMAN biotic model were used to derive concentrations of contamination in the air caused by suspended dust. The following equation was used:

$$C_{air} = (1E-06) R C_{soil} \qquad (6\text{-}1)$$

where

C_{air} = particulate concentration in the air (mg/m^3 or pCi/m^3)

1E-06 = conversion from kg to mg

R = airborne respirable particulate matter (mg/m^3) (measured value of *1.*5E-08 kg/m^3 from PM10 monitoring at the RWMC)

C_{soil} = average soil concentration (mg/kg or pCi/kg).

6.2.2.3 Inhalation of Volatiles. As noted in Section **5.3,** VOC modeling was not included in this ABRA. The IRA results were scaled based on ratios of the inventory modifications identified by Varvel *(2001).* For the IRA, the subsurface model TETRAD computed the flux through the ground surface of any of the volatile contaminants. The volatile flux is the result of vapor-phase diffusion and barometric pumping effects. The flux was input into a "box model" to compute the air concentration used to assess the risk from inhalation of volatiles. The equation used to represent the air concentration resulting from the flux of volatile contaminants is the following:

$$C_{air} = \frac{FLX}{MH \times W \times WS} \times CF1 \times CF2 \qquad (6\text{-}2)$$

where

C_{air} = the air concentration (mg/m^3 or pCi/m^3)

FLX = volatile flux (kg/day or pCi/day)

MH = mixing height (2 m)

W = facility width (181 m)

WS = windspeed (2.83 m/second)

CF1 = conversion factor (1 day/86,400 seconds)

CF2 = conversion factor (1E+06 mg/kg or 1 pCi/pCi).

6.2.2.4 External Radiation Exposure. Exposure to ionizing radiation is caused by concentrations in the surface soil. The average surface concentrations predicted by the biotic model DOSTOMAN were used to estimate the potential exposure.

6.2.2.5 Dermal Absorption from Organic Contaminants in Soil. As noted in Section 5.3, VOC modeling was not included in this ABRA. The IRA results were scaled based on ratios of the inventory revisions. For the IRA, concentrations of organic contaminants in the soil were computed directly by the TETRAD model and used in the exposure calculations. The largest VOC concentration in any grid in the SDA was used for the total SDA risk calculation.

6.2.2.6 Residential Groundwater Ingestion. TETRAD was used to estimate aquifer concentrations anywhere in the modeling domain. Maximum predicted groundwater concentrations along the INEEL boundary were used to quantify potential exposure to contaminated groundwater during the simulated 100-year institutional control period. Estimated groundwater concentrations concurrent with maximum groundwater risk for all contaminants were used to quantify potential exposure for the hypothetical future residential scenario.

6.2.2.7 Residential Ingestion of Homegrown Produce. Concentrations of contaminants in homegrown produce were computed using both the soil concentrations and groundwater concentrations. Groundwater concentrations were used to mimic irrigating produce with contaminated groundwater. The methodology for determining crop concentrations is detailed in an INEEL report on the food-crop-ingestion exposure route (LMITCO 1996).

6.2.2.8 Residential Dermal Absorption of Contaminants in Groundwater. Contaminant concentrations predicted by the subsurface model were used directly to address the dermal exposure to contaminated groundwater.

6.2.2.9 Residential Inhalation of Volatiles from Indoor Use of Groundwater. As noted in Section 5.3, VOC modeling was not included in this ABRA. The IRA results were scaled based on the ratios of the inventory revisions. For the IRA, concentrations of contaminants in the indoor air from indoor water use were given by the following equation:

$$C_{air} = C_{water}\ VF \qquad (6\text{-}3)$$

where

C_{air} = concentration in air (mg/m^3)

C_{water} = concentration in water (mg/L)

VF = volatilization factor (EPA value of 0.5 L/m^3 [EPA 19911).

Maximum soil and groundwater concentrations and the years of the predicted occurrences of maximum concentrations are presented in Table 6-1. Figures illustrating surface soil concentrations used in the risk calculations for each of seven groups of contaminants appear in Section 6.4.3. Predicted groundwater concentrations also are illustrated in Section 6.4.3.

6.2.3 Quantification of Exposure

Contaminant intake is dependent on the ingestion or contact rate with the contaminated media. For radioactive contaminants, exposure was described as a total lifetime intake (in pCi). For hazardous contaminants, exposure was quantified using an intake rate (in mgkg-day). The following sections present methods used to compute intake for each human health exposure pathway.

6.2.3.1 ***Soil Ingestion.*** The equation below was used to compute intake from soil ingestion. For radionuclides, the denominator (BW x AT) is removed from the equation. Default values were taken from Track 2 guidance (DOE-ID 1994).

$$Intake = \frac{C_{soil} \times CF \times IR \times EF \times ED}{BW \times AT} \qquad (6\text{-}4)$$

where

Intake = contaminant intake (mgkg-day or pCi)

and

		Parameter	Occupational Exposure Value	Residential Exposure Value
C_{soil}	=	Contaminant concentration in the soil (mgkg or pCi/g)	Contaminant dependent	Contaminant dependent
CF	=	Conversion factor	10^{-6} kg/mg nonradionuclide or 10^{-3} g/mg radionuclide	10^{-6} kg/mg nonradionuclide or 10^{-3} g/mg radionuclide
IR	=	Ingestion rate of soil (mg/day)	50	100
EF	=	Exposure frequency (day/year)	250	350
ED	=	Exposure duration (year)	25	30
BW	=	Body weight (kg)	70	70
AT	=	Averaging time (day)	9.13E+03 (noncarcinogenic) 2.55E+04 (carcinogenic)	1.10E+04 (noncarcinogenic) 2.55E+04 (carcinogenic).

Table 6-1. Maximum soil and groundwater concentrations for each quantitatively evaluated contaminant of potential concern and associated decay chain members.

Contaminant	Maximum Soil Concentration (pCi/g or mg/kg)[a]	Year of Predicted Maximum Soil Concentration	Maximum Groundwater Concentration (pCi/L or mg/L)[b]	Year of Predicted Maximum Groundwater Concentration
Ac-227	1.95E-06	2274	2.58E-01	3010
Am-241	2.18E+01	2954	1.15E-03	3010
Am-243	2.07E-03	2997	7.71E-07	3010
C-14	2.94E-02	2211	2.02E+04	2282
C1-36	6.33E-05	1963	9.74E+01	2110
Cs-137	7.26E-02	2106	NA	NA
1-129	3.98E-09	1970	2.33E+01	2110
Nb-94	4.63E-01	3010	6.22E-08	3010
Np-237	4.36E-03	2669	2.82E+02	3010
Pa-231	1.21E-06	2234	8.47E-02	3010
Pb-210	1.91E-02	3010	1.96E-01	3010
Pu-238	1.91E-03	2287	1.13E-19	3010
Pu-239	4.0E+00	3010	3.20E-12	3010
Pu-240	4.21E+00	3010	1.70E-15	3010
Ra-226	1.1OE-02	3010	2.32E-02	3010
Sr-90	9.35E+00	2033	NA	NA
Tc-99	3.95E-01	1971	5.23E+03	3010
Th-229	7.35E-05	3010	3.61E-02	3010
Th-230	9.12E-04	3003	3.80E-01	3010
Th-232	9.16E-04	3009	1.97E-07	3010
U-233	1.63E-04	2968	1.71E+01	3010
U-234	8.65E-03	3003	1.06E+03	3010
U-235	6.29E-04	2206	7.94E+01	2663
U-236	5.49E-04	2217	7.09E+01	3010
U-238	1.27E-02	2231	1.62E+03	3010
Carbon tetrachloride	5.94E+00	1967	1.09E+00	2106
Methylene chloride	8.05E-03	1967	2.44E-01	2187
Nitrates	1.51E-02	1999	6.03E+01	2110
Tetrachloroethylene	9.40E-01	1968	2.54E-01	2138

a. Units are pCi/g for radionuclides and mg/kg for nonradionuclides.

b. Units are pCi/L for radionuclides and mg/L for nonradionuclides

6.2.3.2 Inhalation of Fugitive Dust Intake from inhalation can be computed similarly to intake from soil ingestion. That is, the contaminant air concentration was adjusted by factors to account for the type of exposure (residential or occupational) and was compared to the unit risk concentration. The equation below was used to compute intake from inhalation. For radionuclides, the denominator (BW x AT) is removed from the equation. The default values were taken from Track 2 guidance (DOE-ID 1994).

$$Intake = \frac{C_{air} \times IR \times EF \times ED}{BW \times AT} \qquad (6\text{-}5)$$

where

Intake = contaminant intake (mg/kg-day or pCi)

and

Parameter		Occupational Exposure Value	Residential Exposure Value
C_{air}	= Contaminant concentration in the air (mg/m^3 or pCi/m^3)	Contaminant dependent	Contaminant dependent
IR	= Inhalation rate of air (m^3/day)	20	20
EF	= Exposure frequency (day/year)	250	350
ED	= Exposure duration (year)	25	30
BW	= body weight (kg)	70	70
AT	= Averaging time (day)	9.13E+03 (noncarcinogenic) 2.55E+04 (carcinogenic)	1.10E+04 (noncarcinogenic) 2.55E+04 (carcinogenic).

6.2.3.3 Inhalation of Volatiles. As noted in Section 5.3, VOC modeling was not included in this ABRA. The IRA results were scaled based on the ratios of inventory revisions. For the IRA, the methodology and parameter values for computing the intake from inhalation of volatiles is the same as the method used for computing the intake from inhalation of fugitive dust.

6.2.3.4 External Radiation Exposure. The equation below was used to compute total exposure for radionuclides. The parameters and their values are given above. Default values were taken from Track 2 guidance (DOE-ID 1994).

$$Exposure = C_{soil} \times ET \times EF \times ED \times CF \qquad (6\text{-}6)$$

where

Exposure = contaminant intake (pCi-year/g)

and

Parameter		Occupational Exposure Value	Residential Exposure Value
C_{soil}	= contaminant concentration in the soil (pCi/g)	Contaminant dependent	Contaminant dependent
ET	= exposure time (hours/day)	8	24
EF	= exposure frequency (days/year)	250	350
ED	= exposure duration (years)	25	30
CF	= conversion factor	1.14E-04 year/hour	1.14E-04 yearhour.

6.2.3.5 ***Dermal Absorption of Organic Contaminants from Soil.*** As noted in Section 5.3, VOC modeling was not included in this ABRA. The IRA results were scaled based on the ratios of inventory revisions. For the IRA, the absorbed dose of a contaminant is computed based on the methodology for the dermal exposure route EPA. Toxicity values provided in the Integrated Risk Information System (IRIS) database and other EPA sources are developed for the ingestion exposure route. Toxicity values are based on the amount of contaminant ingested, not the amount that actually enters the bloodstream. Only some fraction of the contaminant is absorbed through the gastrointestinal tract after being ingested. The fraction absorbed through the gastrointestinal tract can be used to modify the oral toxicity for use in the dermal exposure route. For organic contaminants, the fraction absorbed through the gastrointestinal tract is large, and is conservatively assumed to be in unity in this analysis. No scaling of the toxicity or intake is required.

The absorbed dose for dermal contact with contaminated soil is computed using the equation below. The default values were taken from EPA Region 10 guidance (EPA 1991).

$$AD = \frac{CS \times CF \times SA \times AF \times ABS \times EF \times ED}{BW \times AT} \qquad (6\text{-}7)$$

where

AD = adsorbed dose (mgkg-day)

and

Parameter		Occupational Exposure Value	Residential Exposure Value
CS	= contaminant concentration in the soil (mgkg)	Contaminant dependent	Contaminant dependent
CF	= conversion factor	10^{-6} kg/mg	10^{-6} kg/mg
SA	= skin surface area (cm^2/event)	5,000	5,000
A F	= soil to skin adherence factor (mg/cm^2)	1	1
ABS	= absorption factor (unitless)	Contaminant dependent	Contaminant dependent
EF	= exposure frequency (events/year)	250	350
ED	= exposure duration (years)	25	30
BW	= body weight (kg)	70	70
AT	= averaging time (day)	9.13E+03 (noncarcinogenic) 2.55E+04 (carcinogenic)	1.10E+04 (noncarcinogenic) 2.55E+04 (carcinogenic).

6.2.3.6 *Residential Groundwater Ingestion.*

The intake from groundwater ingestion was given by the equation below. For radionuclides, the denominator (BW x AT) is removed from the equation. Default values were taken from Track 2 guidance (DOE-ID 1994).

$$Intake = \frac{C_{GW} \times IR \times EF \times ED}{BW \times AT} \quad (6\text{-}8)$$

where

Intake = contaminant intake (mgkg-day or pCi)

and

Parameter			Occupational Exposure Value	Residential Exposure Value
C_{GW}	=	contaminant concentration in groundwater (mg/L or pCi/L)	NA	Contaminant dependent
IR	=	ingestion rate of groundwater (L/day)	NA	2
EF	=	Exposure frequency (days/year)	NA	350
ED	=	Exposure duration (years)	NA	30
BW	=	body weight (kg)	NA	70
AT	=	averaging time (day)	NA	1.10E+04 (noncarcinogenic) 2.55E+04 (carcinogenic).

6.2.3.7 *Residential Ingestion of Homegrown Produce.*

The intake from ingestion of homegrown produce is given by the equation below. For radionuclides, the denominator (BW x AT) is removed from the equation. Default values were taken from Track 2 guidance (DOE-ID 1994). Derivation of the ingestion rates is available in an INEEL report on the food-crop-ingestion exposure route (LMITCO 1996).

$$\text{Intake} = \frac{C_{\text{Produce}} \times \text{IR} \times \text{EF} \times \text{ED} \times \text{CF}}{\text{BW} \times \text{AT}} \quad (6\text{-}9)$$

where

Intake = contaminant intake (mgkg-day or pCi)

and

Parameter		Occupational Exposure Value	Residential Exposure Value
$C_{produce}$	= contaminant concentration in produce (mg/kg or pCi/g)	NA	.Contaminantdependent
IR	= ingestion rate of produce (g/day)	NA	2.76E–01 g/kg-d (nonradionuclide) 1.67E–01 g/d (radionuclide)
EF	= exposure frequency (days/year)	NA	350
ED	= exposure duration (years)	NA	30
CF	= conversion factor (kg/g)	NA	10^{-3} (nonradionuclide only)
BW	= body weight (kg)	NA	70
AT	= averaging time (day)	NA	1.10E+04 (noncarcinogenic) 2.55E+04 (carcinogenic).

6.2.3.8 ***Residential Dermal Absorption of Organic Contaminants in Groundwater.*** As noted in Section 5.3, VOC modeling was not included in this ABRA. The IRA results were scaled based on the ratios of inventory revisions. For the IRA, dermal adsorption from contact with contaminated groundwater was computed using the equation below. The default values were taken from EPA Region 10 guidance (EPA 1991).

$$AD = \frac{DA_{event} \text{ x } SA \text{ x } EF \times ED}{BW \text{ x } AT} \qquad (6\text{-}10)$$

where

AD = absorbed dose (mag-day)

and

Parameter		Occupational Exposure Value	Residential Exposure Value
DA_{event}	= amount absorbed per event (mg/cm^2-event)	NA	See below
SA	= skin surface area (cm^2)	NA	20,000
EF	= exposure frequency (events/year)	NA	350
ED	= exposure duration (years)	NA	30
BW	= body weight (kg)	NA	70
AT	= averaging time (day)	NA	1.10E+04 (noncarcinogenic) 2.55E+04 (carcinogenic).

The amount absorbed per event is given by the equation below. Default values were taken from EPA Region 10 guidance (EPA 1991). Table 5-8 of the EPA dermal exposure assessment (EPA 1992) provides values of K_p and τ.

$$DA_{event} = 2 \times K_p \times C_{water} \times CF \times \sqrt{\frac{6 \times \tau \times t_{event}}{\pi}} \quad (6\text{-}11)$$

where

DA_{event} = amount absorbed per event (mg/cm^2-event)

and

Parameter		Occupational Exposure Value	Residential Exposure Value
K_p	= permeability coefficient for contaminant through skin (cm/hour)	NA	Contaminant specific
C_{water}	= concentration in the water (mg/L)	NA	Contaminant specific
CF	= conversion factor	NA	1E–03 L/cm^3
τ	= lag time (hour/event)	NA	Contaminant specific
t_{event}	= event time (hour/event)	NA	0.17.

6.2.3.9 Residential Inhalation of Volatiles from Indoor Use of Groundwater. As noted in Section 5.3, VOC modeling was not included in this ABRA. The IRA results were scaled based on the ratios of inventory revisions. For the IRA, the intake from indoor use of groundwater was computed using the same methodology and parameter values as for inhalation of volatiles. The only difference is that indoor air concentration was substituted in the equation for the air concentration.

6.3 Toxicity Profiles for Human Health Contaminants of Potential Concern

A toxicity assessment was conducted to identify the potential adverse effects of WAG 7 COPCs and compile toxicity values for use in the ABRA. A toxicity value is a numerical expression of a substance-dose-response relationship. Reference doses (RfDs) and reference concentrations (RfCs) are used to evaluate noncarcinogenic effects. Unit risk values and slope factors (SFs) apply to carcinogenic effects. Each toxicity value is specific to both a particular substance and to the exposure pathway. The majority of the toxicity values for this assessment were obtained from the EPA IRIS database (EPA 2002) and the Health Effects Assessment Summary Tables (HEAST) (EPA 2001).

Each of 24 WAG 7 human health COPCs is classified as either a chemical or a radionuclide. Potential carcinogenic and noncarcinogenic effects were considered for the four chemical COPCs. Only carcinogenic effects were considered for the 20 radioactive COPCs.

For noncarcinogenic effects, descriptions of critical effects, oral RfDs, inhalation RfCs, and MCLs are shown in Table 6-2. A critical effect, as defined by the EPA (1996), is the first adverse effect of a contaminant or its known precursor that occurs as the dose rate increases—that is, the first observable symptom that results from an exposure.

Table 6-2. Toxicity values for quantitatively evaluated noncarcinogens.

Chemical	Critical Effect	Chronic[a] Oral Reference Dose (mg/kg-day)	Chronic Oral Reference Dose Uptake Factor	Chronic[a] Inhalation Reference Concentration (mg/m^3)	Chronic Inhalation Reference Concentration Uptake Factor	Maximum Concentration Level[b] (mg/L)
Carbon tetrachloride	Liver lesions	7.0E-04	1,000	ND	ND	5.0E-03
Methylene chloride	Liver toxicity	6.0E-02	100	3.0E+00[c]	100	5.0E-03
Nitrate	Early clinical signs of hemoglobin in oxidized state in the blood	1.6E+00	1	ND	ND	1.0E+01[d]
Tetrachloro-ethylene	Liver toxicity in mice, weight gain in rats	1.0E-02	1,000	ND	ND	ND

ND means that no data are available.

a. Values **are from the Integrated Risk Information System database (EPA 2002) except where noted.**

b. The maximum contaminant levels (MCLs) are **obtained from 40 CFR 141.**

c. The **toxicity value is from** the **Health Effects Assessment Summary Tables (EPA 2001).**

d. The **MCL for nitrate is 10 mg/L as nitrogen.**

Weight-of-evidence classes, oral SFs, inhalation unit risk values, and MCLs are used to assess carcinogenic toxicity for chemicals. The EPA has grouped substances to describe carcinogenicity according to the weight of evidence that supports the classification. Groups A, B1, B2, and C are described as follows:

- Group A—Direct evidence is sufficient to classify the substance as a probable human carcinogen
- Group B1—Direct evidence is sufficient of carcinogenesis in animals with some supporting human data to classify the substance as a probable human carcinogen
- Group B2—Evidence is sufficient of carcinogenesis in animals with some human data, but of lesser quality than in Group B1, to classify the substance as a possible human carcinogen
- Group C—Some carcinogenesis in animals and humans is evident, but data are sufficient to assess the probability of carcinogenesis.

All radionuclides are classified as Group A carcinogens.

6.3.1 Chemicals

Carbon tetrachloride, methylene chloride, nitrate, and tetrachloroethylene were evaluated for noncarcinogenic effects based on the availability of toxicity data needed for the risk calculations (see Table 6-2). Carbon tetrachloride and methylene chloride also were evaluated for carcinogenic effects. The EPA weight-of-evidence classification and available oral SFs, inhalation unit risks, and MCLs for chemical carcinogens are provided in Table 6-3. Potential toxic effects associated with the evaluated exposure routes and sources of toxicity values used in the toxicity assessment are described in the following sections.

Table 6-3. Toxicity values for quantitatively evaluated chemical carcinogens.

Chemical	EPA Weight of Evidence"	Oral Slope Factor" $(mg/kg\text{-}d)^{-1}$	Inhalation Unit Risk" $(mg/m^3)^{-1}$	MCL[b] (mg/L)
Carbon tetrachloride	B2	1.3E-01	1.5E-05	5.0E-03
Methylene chloride	B2	7.5E-03	4.7E-07	5.0E-03
Tetrachloroethylene	B2	5.1E-02'	NA	NA

a. Values are from the Integrated Risk Information System database **(EPA *2002).***

b. Maximum contaminant levels **(MCLs)** are from **40** CFR **141.**

c. The value for tetrachloroethylene is under **EPA** review and not available. The same value applied in the **7-08** RI/FS (Duncan, Troutman, and Sondrup **1993)** was used.

6.3.1.1 Carbon Tetrachloride. The critical effect of carbon tetrachloride is liver lesions (EPA 2002). Exposure to high levels of carbon tetrachloride can be fatal. The most immediate harmful effects are to the central nervous system. Other common effects include headaches, dizziness, nausea, and vomiting. In severe cases, stupor, coma, and permanent damage to nerve cells can occur (ATSDR 1989).

The liver is sensitive to carbon tetrachloride. Liver damage can result from either acute or chronic exposure. In mild exposure cases, the liver becomes swollen and tender, and fat tends to build up inside the tissue. In severe cases, many cells may be killed, leading to decreased liver function. Carbon tetrachloride can be absorbed through the skin in sufficient quantity to cause liver damage (ATSDR 1989).

The kidneys also are sensitive to carbon tetrachloride. Kidney disease and inflammation leading to kidney failure and death are common effects in humans following inhalation exposure. Abnormally high serous fluid in the lungs (i.e., pulmonary edema) commonly occurs in humans exposed to high levels of carbon tetrachloride in air. Ingestion of carbon tetrachloride has been associated with decreased function of the central nervous system, kidney and lung injury, and marked hepatoxicity.

The occurrence of liver cancer in individuals exposed to carbon tetrachloride fumes, both acutely and for long periods, has been noted in some reports. A number of studies have shown that prolonged administration of high levels of carbon tetrachloride by oral or subcutaneous routes can induce liver tumors in rats, mice, and hamsters (ATSDR 1989). Though no studies have established that inhalation exposure to carbon tetrachloride poses a risk of cancer, the evidence for liver carcinogenicity has been shown by oral or parenteral exposure in animals. Because similar noncarcinogenic effects are observed in the liver following oral and inhalation exposure, it is likely that carcinogenic effects are similar for both types of exposure (i.e., inhalation exposure could lead to liver cancer) (ATSDR 1989).

The EPA has classified carbon tetrachloride as a B2 human carcinogen for both ingestion and inhalation (EPA 1996). The oral SF for carbon tetrachloride is 1.3E-01 $(mg/kg\text{-}day)^{-1}$, and the inhalation unit risk is 1.5E-05 $(mg/m^3)^{-1}$ (EPA 2002). The inhalation SF assumes 40% absorption of carbon tetrachloride. Though tumor incidence and death are indicated in several studies, all the studies are deficient in some respect. Therefore, confidence in the carcinogenic toxicity values is medium.

The evaluation of noncarcinogenic effects after ingestion of carbon tetrachloride is based on an EPA-established chronic RfD, 7.0E-04 mg/kg-d (EPA 2002). The potential for noncarcinogenic effects from inhalation exposure to carbon tetrachloride is not evaluated because of a lack of appropriate data with which to develop a RfC.

6.3.7.2 *Methylene Chloride.* The critical effect of methylene chloride is liver toxicity (EPA 2002). The principal route of human exposure to methylene chloride is inhalation. Evaluation of pulmonary uptake in humans indicates that 70 to 75% of inhaled methylene chloride vapor is absorbed. As for absorption of other lipophilic organic vapors, methylene chloride absorption appears to be influenced by factors other than the vapor concentration. Increased physical activity and higher body fat increases the amount of methylene absorbed by the body (ATSDR 1993).

Effects from inhalation of methylene chloride include headaches, giddiness, stupors, irritability, numbness, and tingling in the limbs. Irritation to the eyes and upper respiratory passages occurs at higher doses. In severe cases, toxic brain disease with hallucinations and effusion of fluid into the alveoli and interstitial spaces of the lungs, coma, and death have been observed. Cardiac arrhythmias have been produced in animals but have not been common in humans. Exposure to methylene chloride may cause elevated carboxyhemoglobin levels that may be significant in smokers, workers with anemia or heart disease, and those exposed to carbon monoxide (Sittig 1985).

The central nervous system is affected adversely in humans and animals at exposure levels of 500 ppm or higher. Noted effects from these exposure levels were decreased visual and auditory functions; however, these effects were reversible once exposure ceased. Similarly, psychomotor performance (reaction time, hand precision, and steadiness) was impaired and alterations in visually evoked response have been observed in humans exposed to higher levels of methylene chloride (ATSDR 1993c).

The EPA has classified methylene chloride as a B2 human carcinogen for both ingestion and inhalation (EPA 2002). The oral SF for methylene chloride is 7.5E-03 $(mg/kg\text{-}day)^{-1}$, and the inhalation unit risk is 4.7E-07 $(mg/m^3)^{-1}$ (EPA 2002). Though important uncertainties remain about pharmacokinetics, pharmacodynamics, and mechanisms of carcinogenicity for methylene chloride, the confidence in the toxicity values is medium.

Evaluation of noncarcinogenic effects after ingestion of methylene chloride is based on a chronic RfD of 6.0E-02 mg/kg-day (EPA 2002). The inhalation RfC for methylene chloride is 3.0E+00 mg/m^3 (EPA 1995). The uncertainty factor of 100 accounts for both expected intraspecies and interspecies variability to the toxicity of this chemical in lieu of specific data. Overall confidence in the oral RfD is medium, because the associated database is rated medium to low based on the limited number of studies.

6.3.1.3 Nitrate. The critical effect of nitrate is early clinical signs of the presence of hemoglobin in an oxidized state in the blood (EPA 2002). Because nitrates can have adverse effects, sodium and potassium nitrate are evaluated for noncarcinogenic effects. The nitrate form of nitrogen is of concern because the ion is highly soluble in water, which enhances leaching, diffusion, and environmental mobility in soil and water.

Nitrates in the environment are of primary concern because they can reduce to nitrates in biological systems. Nitrite is formed from nitrate by certain microorganisms in the alimentary tract and in soil, water, and sewage (Amdur, Doull, and Klassen 1991). Nitrate reduction to nitrite can occur under certain conditions in the stomach as well as in the saliva. Nitrite acts in the blood to oxidize hemoglobin to methemoglobin, which cannot conduct oxygen to the tissues. This condition, known as methemoglobinemia, is caused in humans by high levels of nitrite or, indirectly, excessive levels of nitrate. Nitrate toxicity can result from ingestion of water and vegetables high in nitrates (EPA 2002). Newborns (0 to 3 months) are more susceptible to nitrate toxicity than adults. The increased susceptibility of newborns has been attributed to a high intake per unit weight, the presence of nitrate-reducing bacteria in the upper gastrointestinal tract, condition of the mucosa, and the greater ease of oxidation of fetal hemoglobin.

Other effects associated with the ingestion of nitrates can include hypotension, relatively rapid heartbeat, respiratory dysfunction (from methemoglobinemia), headache, nausea, vomiting, and diarrhea. It has been reported that exposure to nitrates has resulted in convulsions following severe intoxication.

Little scientific basis supports conclusions about the relationship between nitrate concentrations and the carcinogenic potential (EPA 2002). The EPA does not classify nitrates as carcinogens. Therefore, nitrates are not evaluated for carcinogenic effects for WAG 7.

The oral RfD for nitrate is 1.6E+00 mgkg-day (EPA 2002). ***An*** uncertainty factor of **1** was employed because available data define no observable effect levels for the critical toxic effect in the most sensitive human subpopulation. Confidence in the RfD is high, based on evaluation of the database and studies included in the database.

6.3.1.4 Tetrachloroethylene (perchloroethylene). The noted critical effects of tetrachloroethylene are liver toxicity in mice and weight gain in rats (EPA 2002). Exposure to tetrachloroethylene may cause dysfunction of the central nervous system, hepatic injury, and death. Cardiac arrhythmia and renal injury have been observed in animal experiments. Signs and symptoms of exposure to tetrachloroethylene include malaise, dizziness, headaches, increased perspiration, fatigue, difficulty in walking, and slowing of mental ability (Sittig 1985).

Other effects of tetrachloroethylene exposure in humans range from loss of muscular coordination at low concentrations to unconsciousness and respiratory paralysis at high concentrations. Tetrachloroethylene is of moderate to low toxicity by the oral route. Ingestion may cause bleeding and diarrhea and irritate the gastrointestinal membranes. Chronic exposure to tetrachloroethylene most readily affects the central nervous system and liver (ATSDR 1990).

Evaluation of noncarcinogenic effects after ingestion of tetrachloroethylene is based on an RfD of 1.0E-02 mgkg-day (EPA 2002). Tetrachloroethylene was not evaluated for noncarcinogenic effects by inhalation exposure because of a lack of data.

No conclusive evidence has been found to indicate that tetrachloroethylene is carcinogenic in humans. However, animal studies have shown that tetrachloroethylene can cause liver and kidney damage, liver and kidney cancers, and leukemia. Based on evidence from animal studies, tetrachloroethylene is considered carcinogenic in humans (ATSDR 1990). Preliminary toxicity data for carcinogenic effects are available from the EPA (EPA 1989). The oral SF is 5.1E-02 $(mg/kg\text{-}day)^{-1}$, and the inhalation unit risk is 5.8E-07 $(\mu g/m^3)^{-1}$. The value of 5.1E-02 previously was used in the OU 7-08 RI/FS (Duncan, Troutman, and Sondrup 1993) to evaluate the relative impact of tetrachloroethylene on groundwater.

Dermal absorption of tetrachloroethylene is relatively insignificant by comparison to the inhalation exposure route. However, two cases occurred in which workers at a dry-cleaning business had blistering of the skin after accidental exposure (ATSDR 1990).

6.3.2 Radionuclides

The EPA has classified all radionuclides as Group A carcinogens based on the extensive weight of evidence provided by epidemiological studies of radiation-induced cancers in humans (EPA 1995). Target organs for radiation-induced cancers in humans can include the thyroid, breast, lungs, blood (bone marrow), stomach, liver, small and large intestines, brain, bone, esophagus, bladder, pancreas, lymphatic tissue, skin, pharynx, uterus, ovaries, and kidneys (EPA 1989). Any dose of radiation is assumed to produce adverse effects with no minimum threshold for radiation carcinogenesis.

The degree of radiotoxicity associated with a specific radioisotope is dependent on the type of emission (i.e., alpha, beta, or gamma), magnitude of energy, half-life, and exposure pathway. The SFs developed by the EPA reflect those characteristics. Shleien (1992) also grouped nuclides according to toxicity, based on the same characteristics, and rated them from one to four, describing very high, high, moderate, or low radiotoxicity. The 25 radioisotopes evaluated for carcinogenic effects in the ABRA are listed in Table 6-4. The primary decay mode, toxicity classification, pathway-specific SFs, and MCLs are tabulated for each nuclide. Pathway-specific SFs have been identified for ingestion, inhalation, and external exposure. The EPA recently updated their slope factor methodology to include individual ingestion slope factors for water, food, and soil ingestion. The ABRA incorporates this new methodology in the analysis.

Descriptions of bodily effects for specific isotopes are available for only a few radionuclides. Others are assessed in general terms according to the type of decay emission and its associated linear energy transfer value. The linear energy transfer value is "...a measure of the ability of biological material to absorb ionizing radiation; specifically, for charged particles traversing a medium, the energy lost per unit length of path as a result of those collisions with electrons in which the energy lost is less than a specified maximum value..." (Shleien 1992).

Low linear energy transfer isotopes typically are sparsely ionizing gamma or beta radiations and tend to travel farther into tissues than alpha particles. Target organs for low linear energy transfer induced cancers in humans can include the thyroid, breast, and blood (bone marrow) (NCRP 1980). Alpha-emitting isotopes usually exhibit high linear energy transfer and effects tend to be more localized, reflecting the lesser degree of penetration associated with alpha particles.

Consequently, alpha-emitters and low-energy beta particles generally are considered ingestion and inhalation hazards but not a significant external exposure concern. Conversely, gamma radiation can generate significant exposures by inhalation, ingestion, and external exposure. Target organs for gamma-induced cancers in humans can include the thyroid, breast, lung, blood (bone marrow), stomach, liver, small and large intestines, brain, bone, esophagus, bladder, pancreas, lymphatic tissues, skin, pharynx, uterus, ovaries, and kidneys. Breast cancer typically occurs 10 years after exposure (BEIR IV 1988), and thyroid cancer is a late consequence of ionizing radiation.

The most likely tissues to exhibit adverse health effects following intake of transuranic isotopes (i.e., elements of atomic number greater than 92) are the lungs, liver, bone (bone marrow), and lymph nodes, and to a lesser degree thyroid gland, gonads, and kidneys (BEIR IV 1988). By far the greatest emphasis has been placed on the lungs and bone because these two tissues have been the predominant sites of neoplasia in experimental animals.

The EPA SFs reflect the considerations discussed above. Additional descriptions available for specific radioactive elements are given below.

6.3.2.1 Actinium. Data from early studies have shown the absorption of actinium through the gastrointestinal tract to be very low. Like other actinides, intravenously or intramuscularly injected actinium becomes concentrated in the liver, skeleton, and to some extent, the kidneys (ICRP 1978).

6.3.2.2 ***Americium.*** Data from animal studies have shown the absorption of americium through the gastrointestinal tract to be very low, Americium compounds are more rapidly cleared from the lung than are compounds of plutonium (ICRP 1978). After inhalation, Am-241 resides more in the skeleton than in the lungs (BEIR IV 1988), and approximately 30% of inhaled Am-241 resides in the liver. Inhalation has been shown to induce lung tumors in rats (BEIR IV 1988).

Table 6-4. Slope factors and other data used to estimate carcinogenic risks for radionuclide contaminants of potential concern and associated decay chain members.

Radionuclide	Primary Decay Mode[a]	Toxicity Group[b]	Water Ingestion Slope Factor[c] $(pCi)^{-1}$	Food Ingestion Slope Factor[c] $(pCi)^{-1}$	Soil Ingestion Slope Factor[c] $(pCi)^{-1}$	Inhalation Slope Factor[c] $(pCi)^{-1}$	External Exposure Slope Factor[c] $(y/pCi/g)^{-1}$	MCL[d] (pCi/L)
Ac-227+D	B, A	1	4.86E-10	6.53E-10	1.16E-09	2.09E-07	1.47E-06	1.5E+01
Am-241	A	1	1.04E-10	1.34E-10	2.17E-10	2.81E-08	2.76E-08	1.5E+01
Am-243+D[e]	A	1	1.08E-10	1.42E-10	2.32E-10	2.70E-08	6.36E-7	1.5E+01
C-14	B	3	1.55E-12	2.00E-12	2.79E-12	7.07E-12	7.83E-12	2.00E+03
Cl-36	B[f]	2	3.30E-12	4.44E-12	7.66E-12	2.50E-11	1.74E-09	7.00E+02
Cs-137+D[e]	B	3	3.04E-11	3.74E-11	4.33E-11	1.19E-11	2.55E-06	2.00E+02
I-129	B	4	1.48E-10	3.22E-10	2.71E-10	1.60E-10	6.10E-09	1.00E+00
Nb-94	B'	2	7.77E-12	1.11E-11	2.05E-11	3.77E-11	7.29E-06	1.07E+03
Np-237+D[e]	A	1	6.74E-11	9.10E-11	1.62E-10	1.77E-08	7.97E-07	1.5E+01
Pa-231	A	1	1.73E-10	2.26E-10	3.74E-10	4.55E-08	1.39E-07	1.50E+01
Pb-210+D[e]	B	1	8.81E-10	3.44E-09	1.84E-09	1.39E-08	4.21E-09	NA
Pu-238	SF, A	1	1.31E-10	1.69E-10	2.72E-10	3.36E-08	7.22E-11	1.50E+01
Pu-239	A	1	1.35E-10	1.74E-10	2.76E-10	3.33E-08	2.00E-11	1.50E+01
Pu-240	SF, A	1	1.35E-10	1.74E-10	2.77E-10	3.33E-08	6.98E-11	1.50E+01
Ra-226+D[e]	A	1	3.86E-10	5.15E-10	7.30E-10	1.16E-08	8.49E-06	5.00E+00
Sr-90+D	B	2	7.40E-11	9.53E-11	1.44E-10	1.13E-10	1.96E-08	8.00E+00
Tc-99	B	3	2.75E-12	4.00E-12	7.66E-12	1.41E-11	8.14E-11	9.00E+02
Th-229+D[e]	A	1	5.28E-10	7.16E-10	1.29E-09	2.25E-07	1.17E-06	1.50E+01
Th-230	A	1	9.10E-11	1.19E-10	2.02E-10	2.85E-07	8.19E-10	1.50E+01
Th-232	A	2	1.01E-10	1.33E-10	2.31E-10	4.33E-08	3.42E-10	1.50E+01
U-233	A	1	7.18E-11	9.69E-11	1.60E-10	1.16E-08	9.82E-10	2.90E+05[g]
U-234	A	1	7.07E-11	9.55E-11	1.58E-10	1.58E-08	2.52E-10	1.87E+05[g]

Table 6-4. **(continued).**

Radionuclide	Primary Decay Mode[a]	Toxicity Group[b]	Water Ingestion Slope Factor[c] $(pCi)^{-1}$	Food Ingestion Slope Factor[c] $(pCi)^{-1}$	Soil Ingestion Slope Factor[c] $(pCi)^{-1}$	Inhalation Slope Factor[c] $(pCi)^{-1}$	External Exposure Slope Factor[c] $(y/pCi/g)^{-1}$	MCL[d] (pCi/L)
U-235+D[e]	A	4	7.18E-11	9.76E-11	1.63E-10	1.01E-08	5.43E-07	6.49E+01[g]
U-236	A	2	6.70E-11	9.03E-11	1.49E-10	1.05E-08	1.25E-10	1.94E+03[g]
U-238+D[e]	SF, A	4	8.71E-11	1.21E-10	2.10E-10	9.35E-09	1.14E-07	1.01E+01[g]

a. Data are taken from Table 8.13 in Shleien (1992) except as noted. Only primary emissions are listed. Many isotopes are characterized by multiple decay modes. Most emit gamma rays.
A = alpha emission
B = beta emission
EC = electron capture
SF = spontaneous fission.

b. Values are taken from Table 11.1.1.1 in Shleien (1992).
Group 1 = very high radiotoxicity
Group 2 = high radiotoxicity
Group 3 = moderate radiotoxicity
Group 4 = low radiotoxicity.

c. Values are taken from the EPA Health Effects Assessment Summary Tables (EPA 2001).

d. Values are taken from 40 CFR 141.

e. +D indicates that slope factors include the effects of daughter products.

f. Data are taken from GE (1989).

g. Based on proposed MCL of 30 µg/L total uranium.

6.3.2.3 Carbon. Carbon is readily absorbed into the bloodstream through the gastrointestinal tract or the lungs and subsequently is deposited throughout all organs and tissues of the body. Data from the International Commission on Radiation Protection (ICRP 1975) suggest that the biological half-life of dietary carbon in the body is about 40 days. However, studies of autopsy samples of people exposed to C-14 from fallout indicate that bone collagen and bone mineral retain carbon with a biological half-life in excess of ***5*** years (ICRP 1978).

6.3.2.4 Cesium. Regardless of the mode of exposure, Cs-137 is rapidly absorbed into the bloodstream and distributes throughout the active tissues of the body. Metabolically, Cs-137 behaves as an analog of potassium. Distribution of cesium throughout the body and energetic beta and gamma radiation from the decay daughter, Ba-137m, result in essentially whole-body irradiation (Amdur, Doull, and Klassen 1991).

6.3.2.5 Iodine. Iodine is absorbed rapidly and almost completely through the gastrointestinal tract, mainly from the small intestine. Approximately 30% of the iodine that enters the blood is retained in the thyroid (ICRP 1978). Iodine eventually is lost from the thyroid gland in the form of organic iodine and is retained in the remaining organs and tissues within the body. The biological half-life of iodine within the body is approximately 120 days (ICRP 1978).

6.3.2.6 Lead. The fractional absorption of lead through the gastrointestinal tract of humans has an estimated range of 0.05 to 0.65 (ICRP 1978). It has been shown that when injected in the body, Pb-210 is deposited in bone, liver, and kidneys but is tenaciously retained only by mineral bone (ICRP 1978).

6.3.2.7 Neptunium. Data from animal studies have shown the absorption of neptunium through the gastrointestinal tract to be very low. Experiments on rats indicate that neptunium is cleared from the lungs more rapidly than plutonium. Data on the distribution and retention of neptunium in rats indicate that the metabolic behavior of neptunium is similar to that of plutonium. However, in the skeleton, the distribution of neptunium may more closely resemble calcium than plutonium (ICRP 1978).

6.3.2.8 Niobium. Data from the ICRP Reference Man report (ICRP 1975) indicate that a large fraction of dietary niobium is absorbed through the gastrointestinal tract. However, other studies on a number of compounds of the element have indicated that the fractional absorption is 0.01 or less in small animals (ICRP 1978). Inhaled niobium oxide is tenaciously retained in the lungs. Animal studies have shown a preferential retention of niobium in mineral bone, with a concentration 10 times the whole-body average, and in the kidneys, spleen, and testes, with concentrations three to five times the whole-body average.

6.3.2.9 Plutonium. After inhalation, plutonium may remain in the lungs but can move to the bones and liver (BEIR V 1990). Plutonium generally stays in the body for a very long time and continues to expose the surrounding tissues to radiation (ATSDR 1990a), increasing the probability of carcinogenesis over time. Approximately 50% of the plutonium that enters the blood is retained in the bone and 30% in the liver with retention times of 20 to 50 years (BEIR IV 1988). Inhalation can cause lung tumors in rats, and dermal absorption is limited (BEIR IV 1988).

Plutonium absorption through the gastrointestinal tract appears to be limited but is increased with decreased iron and calcium levels (BEIR IV 1988). Data have been reported that indicate a much higher gastrointestinal absorption for certain compounds of plutonium that are unlikely to be encountered in occupational exposures, (e.g., hexavalent plutonium compounds, citrates, and other organic complexes). Absorption also is increased in the very young (ICRP 1978).

6.3.2.10 Protactinium. Data from early studies have shown the absorption of protactinium through the gastrointestinal tract to be very low. Protactinium has been shown in animal studies to be deposited primarily in the skeleton, with the liver and kidneys as secondary sites of deposition (ICRP 1978). Protactinium deposited in the skeleton is retained there with a biological half-life in excess of 100 days. Protactinium deposited in the liver or kidneys has a biphasic retention with the two components having biological half-lives of about 10 and **60** days, respectively.

6.3.2.11 Radium. Radium, as a metabolic analog of calcium, is readily absorbed through the gastrointestinal tract or the lungs into the bloodstream and subsequently is deposited in the bones. Values for fractional absorption through the gastrointestinal tract have been observed in a range from 0.15 to 0.21 (ICRP 1978). During the first few days after intake, radium becomes concentrated heavily on bone surfaces, and then gradually shifts its primary deposition site to bone volume. A large percentage of the subjects exposed to high doses of radium have developed bone cancer (BEIR IV 1988).

6.3.2.72 Strontium. Strontium, as a metabolic analog of calcium, is readily absorbed into the bloodstream through the gastrointestinal tract or the lungs and subsequently is deposited in the bones. Observations indicate that a single brief oral, intravenous, or inhalation intake generates a high incidence of tumors in bones and bone-related tissues (BEIR V 1990). Inhalation is the major risk. Data from animal studies indicate that exposure to strontium results in lung and possibly liver damage (Sittig 1985).

6.3.2.13 Technetium. Technetium is readily absorbed through the gastrointestinal tract or the lungs into the bloodstream. Once in the body, technetium subsequently is deposited in the thyroid, gastrointestinal tract, and liver (ICRP 1978).

6.3.2.14 Thorium. Thorium is incorporated into the body mainly by inhalation. It is poorly absorbed through the gastrointestinal tract, and approximately 60% of the thorium body burden is present in the skeleton (BEIR IV 1988). In the body, thorium tends to stay where it is first deposited. When injected into humans as the drug Thorotrast, thorium is deposited in the liver, spleen, bone marrow, and lymph nodes (BEIR IV 1988). Because of its deposition in the bone marrow in which red blood cells are formed, thorium-induced anemia has been observed in conjunction with therapeutically administrated Thorotrast. Liver cancers also have been associated with Thorotrast therapy (BEIR IV 1988).

6.3.2.15 Uranium. Uranium and its compounds are highly toxic. Studies have shown that fractions on the order of 0.005 to 0.05 of a uranium compound are likely to be absorbed into the blood through the gastrointestinal tract (ICRP 1978). Soluble uranium compounds such as UF_6, UO_2F_2, and $UO_2(NO_3)_2$ are absorbed rapidly through the lungs (ICRP 1978). Retention times for uranium in the body may range from **20** to 50 years (ICRP 1978). Major target organs for uranium toxicity are the respiratory system, blood, liver, lymphatic system, kidneys, skin, and bone marrow. Reports have confirmed that carcinogenicity is related to dose and exposure time. Soluble compounds have been reported to cause lung and bone cancers and cancer of the lymphatic tissues, whereas insoluble compounds have been reported to cause cancer of the lymphatic and blood-forming tissues (Sittig 1985).

6.4 Risk Characterization

Risk characterization involves estimating the magnitude of potential adverse human health effects from released COPCs. Specifically, risk characterization combines the results of the exposure and toxicity assessments to develop numerical estimates of the health risk. These estimates, with a given intake, are either comparisons of exposure levels with appropriate RfDs or estimates of the lifetime cancer risk.

6.4.1 Generalized Approach

To quantify human health risks, contaminant intakes are calculated for each COPC for each applicable exposure route. As discussed in Section 6.2, these contaminant intakes are based on the modeled soil and groundwater concentrations listed in Table 6-1. The equations used to estimate the risks for each pathway are discussed below.

6.4.1.1 Carcinogenic Health Effects. The following equations are used to obtain numerical estimates (i.e., probability) of lifetime cancer risks:

$$Risk = Intake \times SF \tag{6-12}$$

where

Risk = potential lifetime cancer risk (unitless)

SF = slope factor, for chemicals $(mg/kg/day)^{-1}$, or radionuclides $(pCi)^{-1}$

Intake= chemical intake rate (mg/kg/day), or total radionuclide intake (pCi).

The linear low-dose equation shown in Equation 6-12 is valid at low risk levels (i.e., below the estimated risk of 1E–02). In accordance with the EPA Risk Assessment Guidance *to Superfund* (RAGS) (EPA 1989), risks that are greater than 1E–02 should be calculated using the one-hit equation. While none of the WAG 7 COPCs fall into this category, the one-hit equation is described below for completeness.

$$Risk = 1 - exp^{(-Intake \times SF)} \tag{6-13}$$

where

Risk = potential lifetime cancer risk (unitless)

SF = slope factor, for chemicals $(mg/kg/day)^{-1}$, or radionuclides $(pCi)^{-1}$

Intake= chemical intake rate (mg/kg/day), or total radionuclide intake (pCi).

To develop the total risk for each contaminant, each pathway risk is summed as follows:

$$Risk, = \Sigma\ Risk_i \tag{6-14}$$

where

$Risk_T$ = total cancer risk for that contaminant

$Risk_i$ = risk for the ith pathway.

Similarly, the total risk for each contaminant is summed to estimate the potential cumulative cancer risk associated with the SDA.

6.4.1.2 Noncarcinogenic Effects. Health risks associated with exposure to individual noncarcinogenic compounds are evaluated by calculating hazard quotients (HQs). The quotient for health hazards is the ratio of intake to the RfD, as follows:

$$HQ = Intake/RfD \tag{6-15}$$

where

HQ = noncarcinogenic HQ (unitless)

Intake = chemical intake rate (mg/kg/day)

RfD = reference dose (mg/kg/day).

Hazard indexes (HIs) are calculated by summing the HQs for each chemical across all exposure routes. If the HI for any COPC exceeds 1.0, potential health effects from exposure to the COPC may be a concern. The contaminant-specific HI is calculated using the following equation:

$$HI = \Sigma HQ \tag{6-16}$$

where

HI = hazard index (unitless)

HQ_i = hazard quotient for each pathway (unitless).

Similarly, the HI estimated for each contaminant as described above can be summed to provide a cumulative HI for the entire SDA.

6.4.2 Estimates of the Potential Human Health Risk

Risk results are summarized in this section. Graphical illustrations of risk results for the hypothetical future residential scenario are found in Section 6.4.3. All exposure pathways were simulated for 1,000 years from 2010 (to the year 3010). Because the groundwater pathway risk did not peak for many of the contaminants during the first 1,000 years, the groundwater pathway was simulated until either a peak was reached or for a total of 10,000 years.

Risks and HIs for the occupational exposure scenario are provided in Table 6-5. Peak risks and HIs through the simulated 100-year institutional control period ending in 2110 and the 1,000-year simulation period ending in 3010 are listed. A comparison of the results in Table 6-5 for the occupational scenarios with the hypothetical future residential risk estimates in Table 6-6 shows that occupational risks are bounded by the residential risks.

The maximum risk and HI for each contaminant for the 1,000-year simulation period are provided in Table 6-6 for hypothetical future residential exposures. The peak risks, the years in which the peak risks occur, and the major pathways contributing to the total risks are also provided in Table 6-6. The peak risk or HI is for the maximum anywhere in the aquifer and is not spatially consistent. That is, a receptor with a well in one location would not see the maximum groundwater risk for all contaminants. The total risk (Figure 6-2) is shown as a spatially consistent receptor. Those contaminants with an HI greater than 1.0 or a risk greater than or equal to 1E-06 are highlighted. The radionuclides C-14, Np-237, Sr-90, Tc-99, U-234, U-235, U236, and U-238, and the chemicals carbon tetrachloride and tetrachloroethylene have peak risk values greater than 1E-04. Plots of the risk for each of the 10 risk drivers greater than 1E-04 are shown in Figures 6-3 through 6-12. Contaminants with a total HI greater than 1.0, carbon tetrachloride, nitrates, and tetrachloroethylene, are illustrated in Figures 6-13, 6-14, and 6-15. Contaminants with a carcinogenic risk in the range of 1E-06 to 1E-04 include the radioisotopes Ac-227, Am-241, C1-36, Cs-137, I-129, Nb-94, Pa-231, Pu-239, Pu-240, Ra-226, and U-233 and the nonradioactive contaminant methylene chloride. Plots for each of these 12 contaminants are included in Figures 6-16 through 6-27.

Table 6-5. Summary of estimated risks and hazard indexes for current and future occupational exposure scenarios.

Contaminant	Peak Risk Through 100-Year Simulation Period	Year of Peak Risk	Peak Risk Through 1,000-Year Simulation Period	Year of Peak Risk
Ac-227	6.E-14	2110	2.E-11	2273
Am-241	9.E-09	2110	5.E-06	2951
Am-243	2.E-11	2110	8.E-09	3005
C-14	7.E-12	2051	2.E-11	2209
C1-36	7.E-13	1963	7.E-13	1963
Cs-137	1E-06	2105	1E-06	2105
I-129	3.E-16	1970	3.E-16	1970
Nb-94	1.E-08	2110	2.E-05	3007
Np-237	3.E-10	2110	2.E-08	2664
Pa-231	5.E-15	2110	1.E-12	2234
Pb-210	6.E-14	2110	4.E-09	3005
Pu-238	1.E-12	2110	2.E-10	2288
Pu-239	5.E-11	2110	4.E-07	3010
Pu-240	5.E-11	2110	4.E-07	3008
Ra-226	2.E-11	2110	5.E-07	3005
Sr-90	1E-06	2033	1E-06	2033
Tc-99	5.E-10	1971	5.E-10	1971
Th-229	4.E-13	2110	5.E-10	3010
Th-230	5.E-15	2110	5.E-10	3005
Th-232	9.E-14	2110	1.E-10	3007
U-233	3.E-14	1997	8.E-12	2972
U-234	1.E-12	2110	5.E-10	3005
U-235	2.E-11	2073	2.E-09	2208
U-236	2.E-13	2110	2.E-11	2217
U-238	6.E-11	2110	9.E-09	2234
Carbon tetrachloride	2E-03	1968	2E-03	1968
Methylene chloride	5E-07	1968	5E-07	1968

Contaminant	Peak Hazard Index Through 100-Year Simulation Period	Year of Peak Risk	Peak Hazard Index Through 1,000-Year Simulation Period	Year of Peak Risk
Carbon tetrachloride	4E-03	1967	4E-03	1967
Methylene chloride	7E-08	1967	7E-08	1967
Nitrate	5E-06	1999	5E-06	1999
Tetrachloroethylene	5E-05	1968	5E-05	1968

Table 6-6. Maximum simulated risks and hazard indexes for the 1,000-year simulation period for a hypothetical future residential exposure scenario.

Contaminant of Potential Concern	Simulation Group	Peak Risk	Year	Peak Hazard Index	Year	Primary Exposure Pathway
Ac-227	2	3.E-06	3010[a]	NA[b]	NA	Groundwater ingestion
Am-241	1	3.E-05	2953	NA	NA	Soil ingestion, inhalation, external exposure, and crop ingestion
Am-243	2	4.E-08	3010[a]	NA	NA	External exposure
C-14	7	6.E-04	2278	NA	NA	Groundwater ingestion
Cl-36	7	6.E-06	2110	NA	NA	Groundwater and crop ingestion
Cs-137	NA	5E-06	2110	NA	NA	External exposure
I-129	7	6.E-05	2110	NA	NA	Groundwater ingestion
Nb-94	7	8.E-05	3010[a]	NA	NA	External exposure
Np-237	1	4.E-04	30108	NA	NA	Groundwater ingestion
Pa-231	2	3.E-06	3010[a]	NA	NA	Groundwater ingestion
Pb-210	4, 5	5.E-07	3010[a]	NA	NA	Groundwater ingestion
Pu-238	4	1.E-09	2286	NA	NA	Soil and crop ingestion
Pu-239	2	2.E-06	3010[a]	NA	NA	Soil and crop ingestion
Pu-240	3	2.E-06	3010[a]	NA	NA	Soil and crop ingestion
Ra-226	4, 5	3.E-06	3010	NA	NA	External exposure
Sr-90	NA	1E-04	2110			Crop ingestion
Tc-99	7	4.E-04	2110	NA	NA	Groundwater ingestion and crop ingestion
Th-229	1	4.E-07	3010'	NA	NA	Groundwater ingestion
Th-230	4, 5	7.E-07	3010[a]	NA	NA	Groundwater ingestion
Th-232	3	1.E-09	3010[a]	NA	NA	Crop ingestion
U-233	1	3.E-05	3010[a]	NA	NA	Groundwater ingestion
U-234	4, 5	2.E-03	3010'	NA[b]	NA[b]	Groundwater ingestion
U-235	2	1.E-04	2662	NA[b]	NA[b]	Groundwater ingestion
U-236	3	1.E-04	3010"	NA[b]	NA[b]	Groundwater ingestion
U-238	5	3.E-03	3016	NA[b]	NA[b]	Groundwater ingestion
Carbon tetrachloride	Scaled IRA	2.E-03	2105	5.E+01	2105	Groundwater ingestion
Methylene chloride	Scaled IRA	2.E-05	2185	1.E-01	2185	Groundwater ingestion
Nitrates	6	NA[b]	NA[b]	1.E+00	2120	Groundwater ingestion
Tetrachloroethylene	Scaled IRA	2E-04[c]	1968	1.E+00	2137	Groundwater ingestion and dermal exposure to contaminated water

Note: For toxicological *risk*, the peak hazard index (HI) is given, and for carcinogenic probability, the peak risk is given.
Red = carcinogenic risk ≥ 1E-04.
Blue = carcinogenic risk ≥ 1E-06 and < 1E-04.
Pink = toxicological (noncarcinogenic) HI ≥ 1.0.

a. The peak risks and HIs for the contaminant did not *occur* before the end of the 1,000-year simulation period. Only groundwater ingestion risks and HIs were simulated for 10,000 years. The risks and HIs for the 10,000-year simulation period are illustrated in Section 6.4.

b. NA = not applicable.

c. The risk estimate is based on a slope factor that is currently under EPA review.

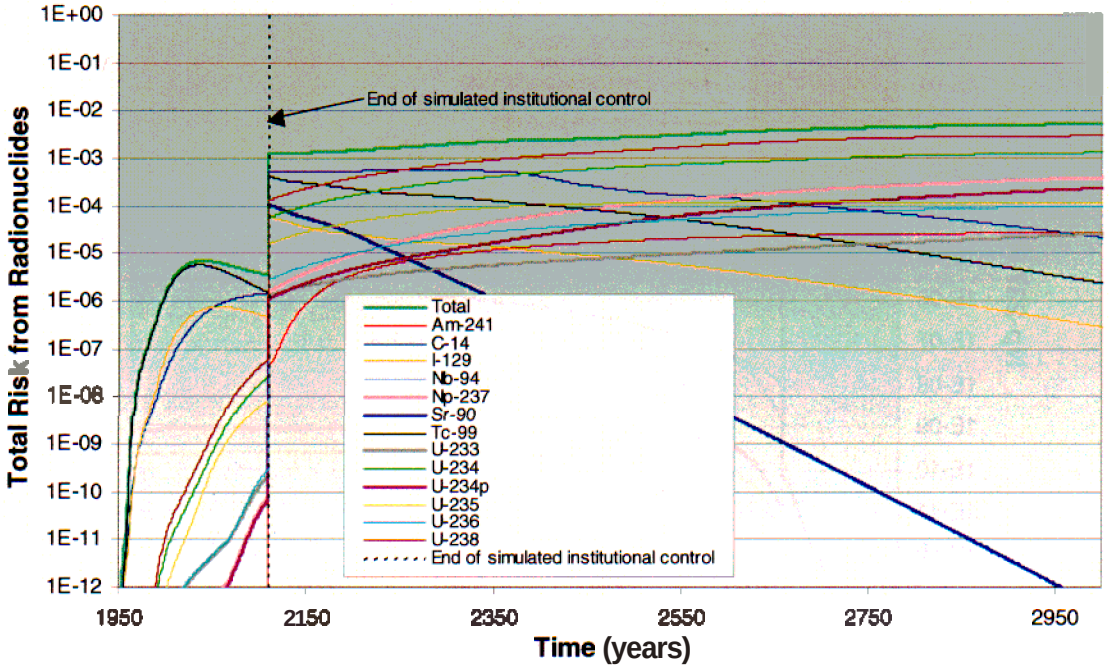

Figure 6-2. Total carcinogenic risks for all radionuclides for hypothetical future residential exposure pathways

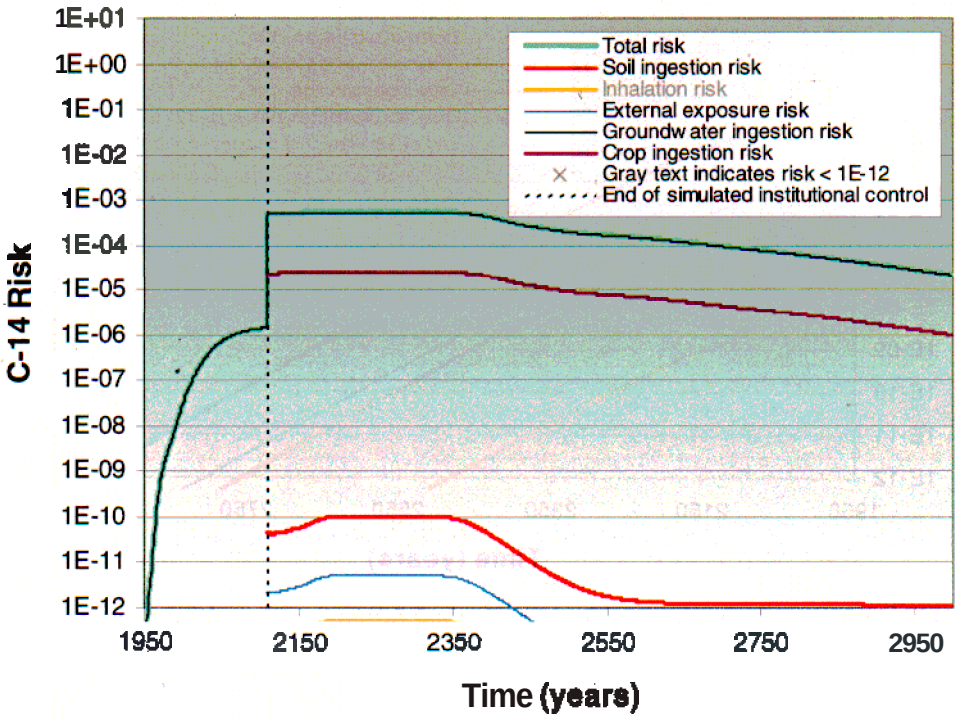

Figure 6-3. Carbon-14 carcinogenic risks for hypothetical future residential scenario exposure pathways,

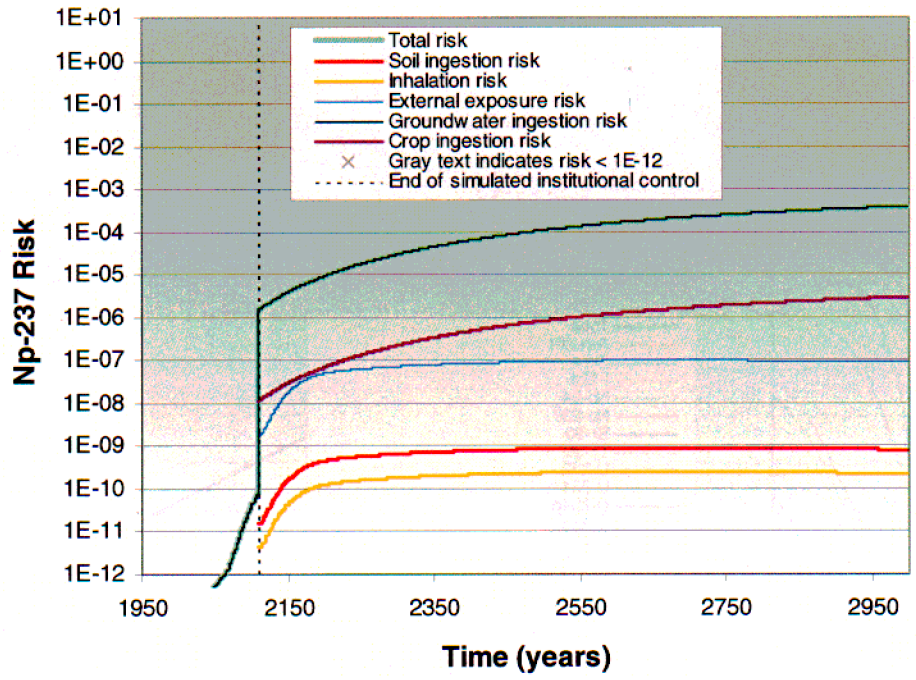

Figure 6-4. Neptunium-237 carcinogenic risks for hypothetical future residential exposure pathways.

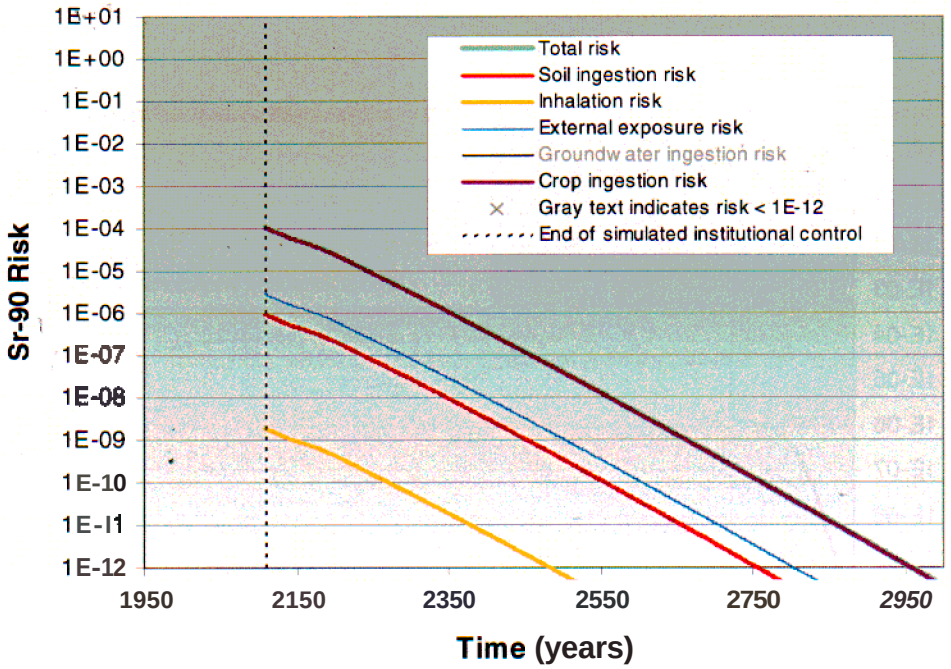

Figure 6-5. Strontium-90 carcinogenic risks for hypothetical future residential exposure pathways.

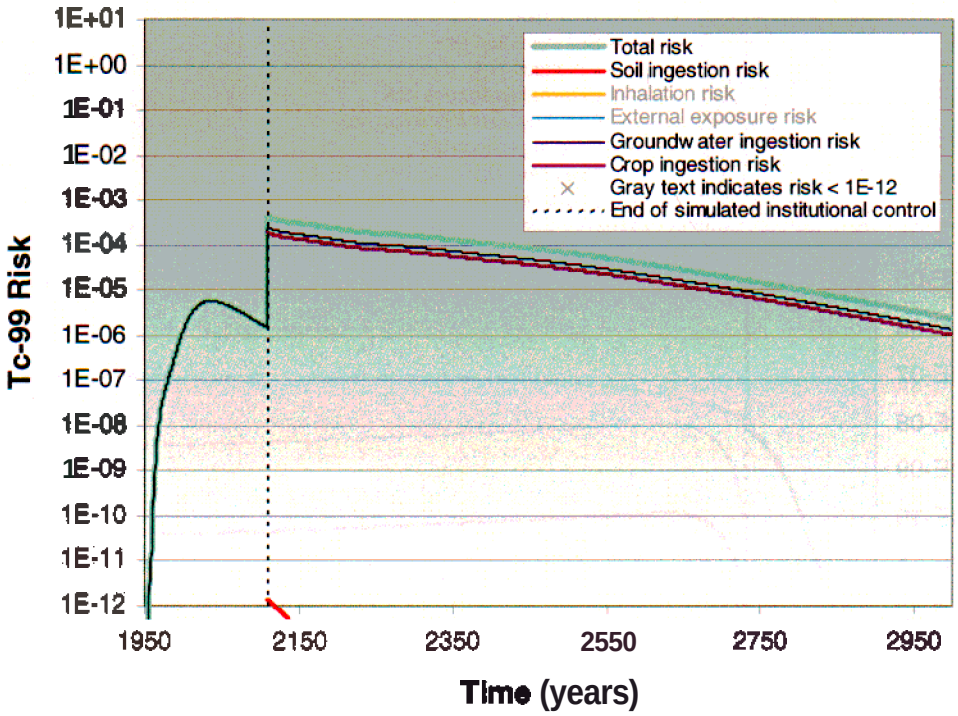

Figure 6-6. Technetium-99 carcinogenic risks for hypothetical future residential exposure pathways.

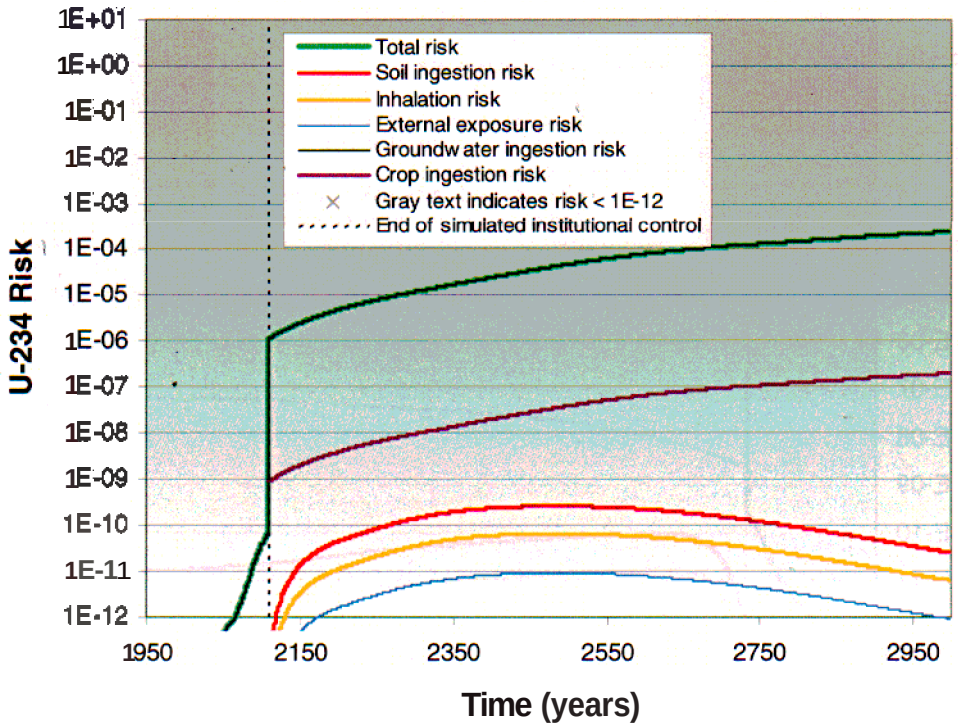

Figure 6-7. Uranium-234 carcinogenic risks for hypothetical future residential exposure pathways.

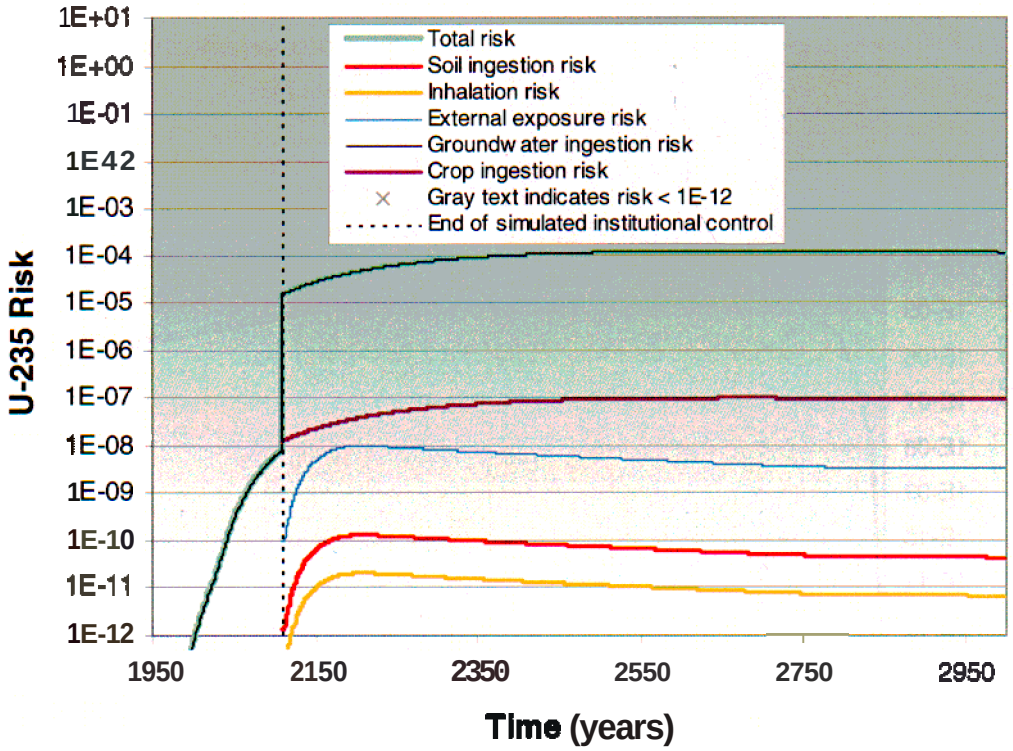

Figure 6-8. Uranium-235 carcinogenic risks for hypothetical future residential exposure pathways.

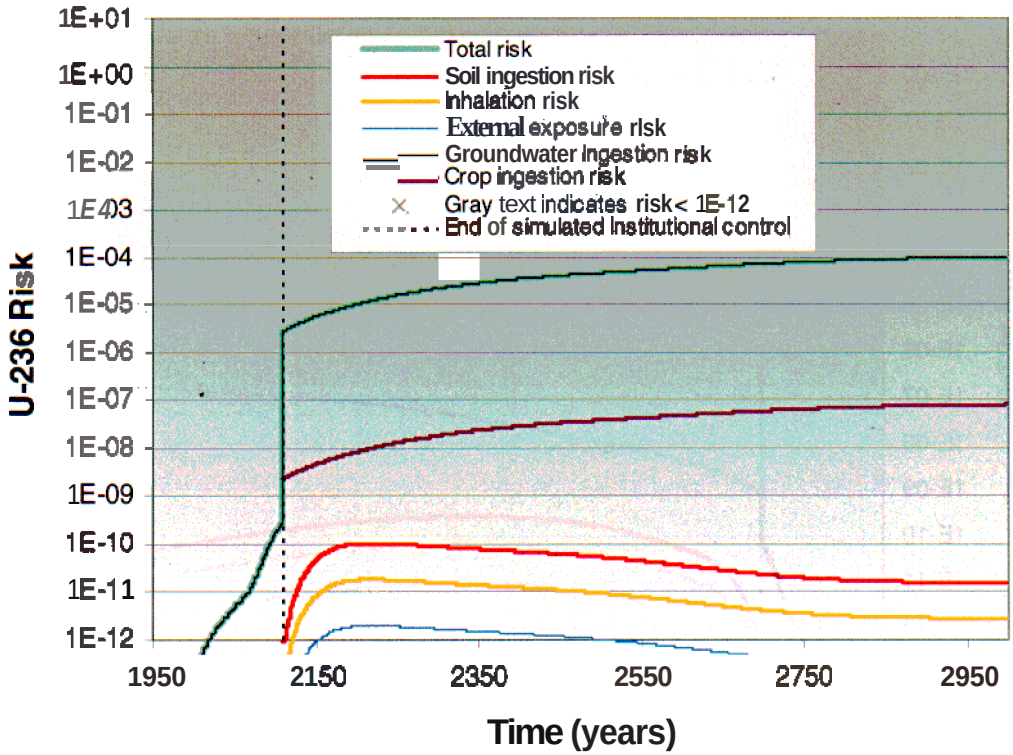

Figure 6-9. Uranium-236 carcinogenic risks for hypothetical future residential exposure pathways.

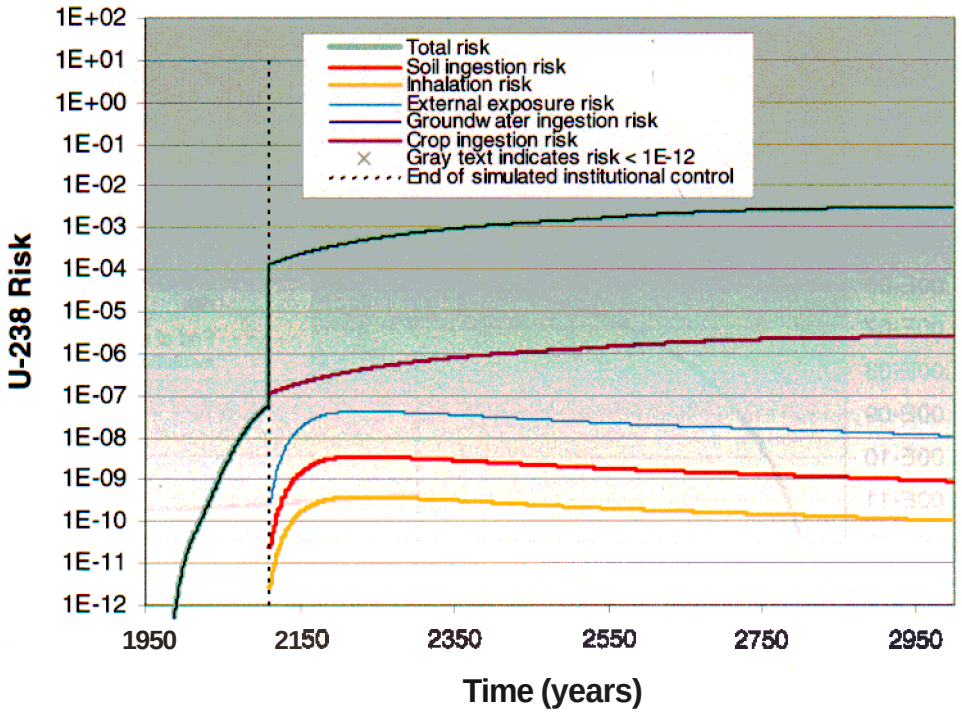

Figure 6-10. Uranium-238 carcinogenic risks for hypothetical future residential exposure pathways;

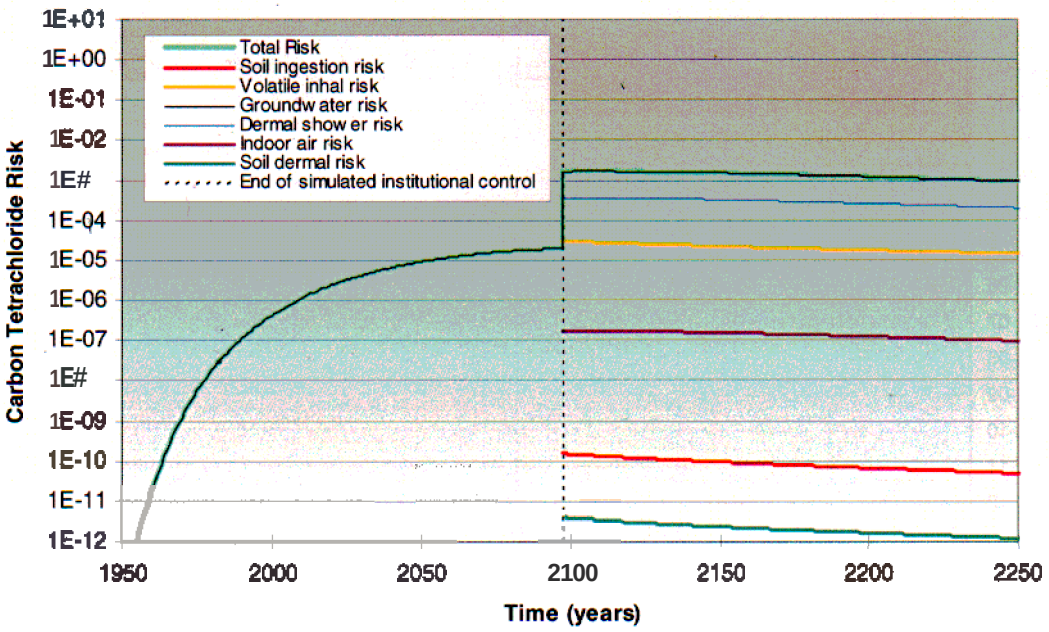

Figure 6-11. Carbon tetrachloride carcinogenic risks for hypothetical future residential **exposure pathways.**

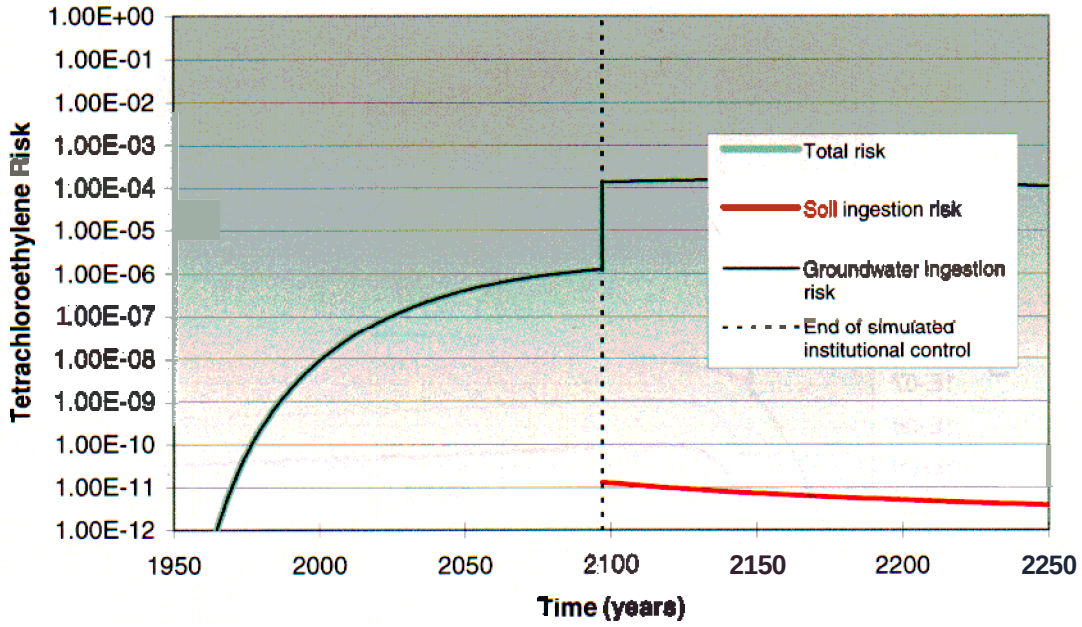

Figure 6-12. Tetrachloroethylene carcinogenic risks for hypothetical future residential exposure pathways,

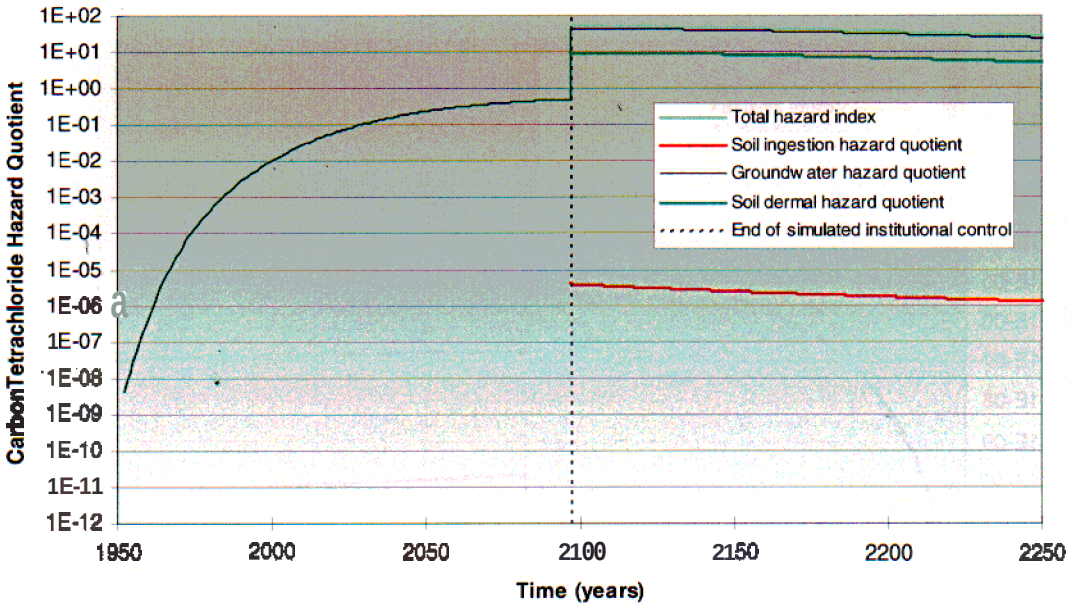

Figure **6-13. Carbon tetrachloride hazard index for hypothetical future residential exposure pathways.**

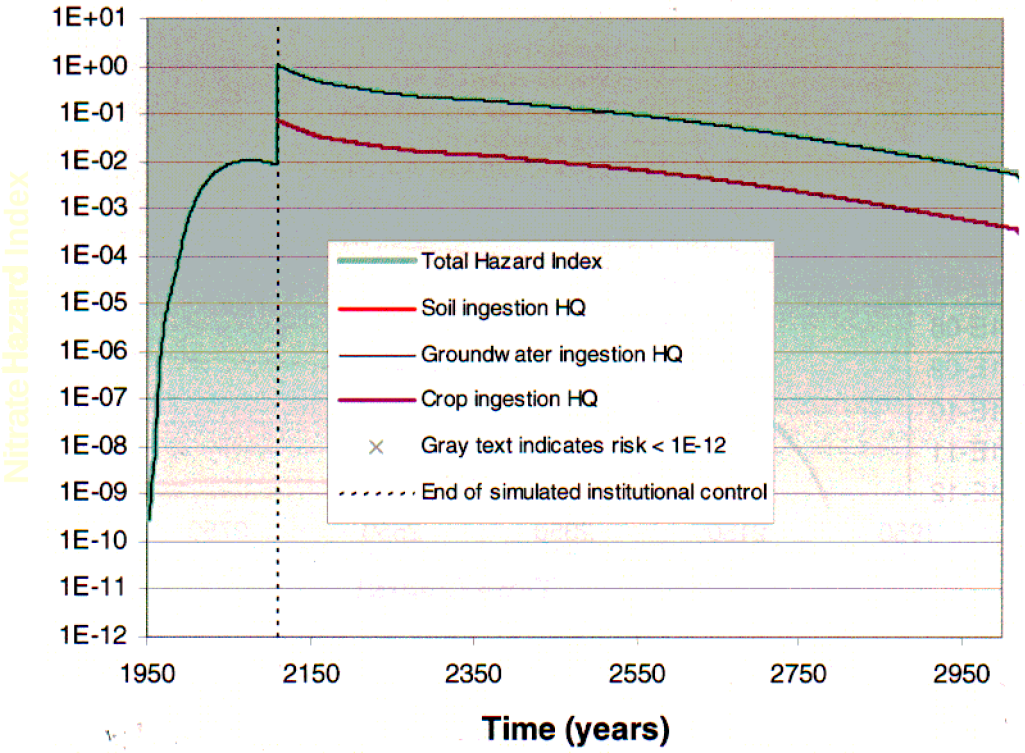

Figure 6-14. Nitrate hazard index for hypothetical future residential exposure pathways.

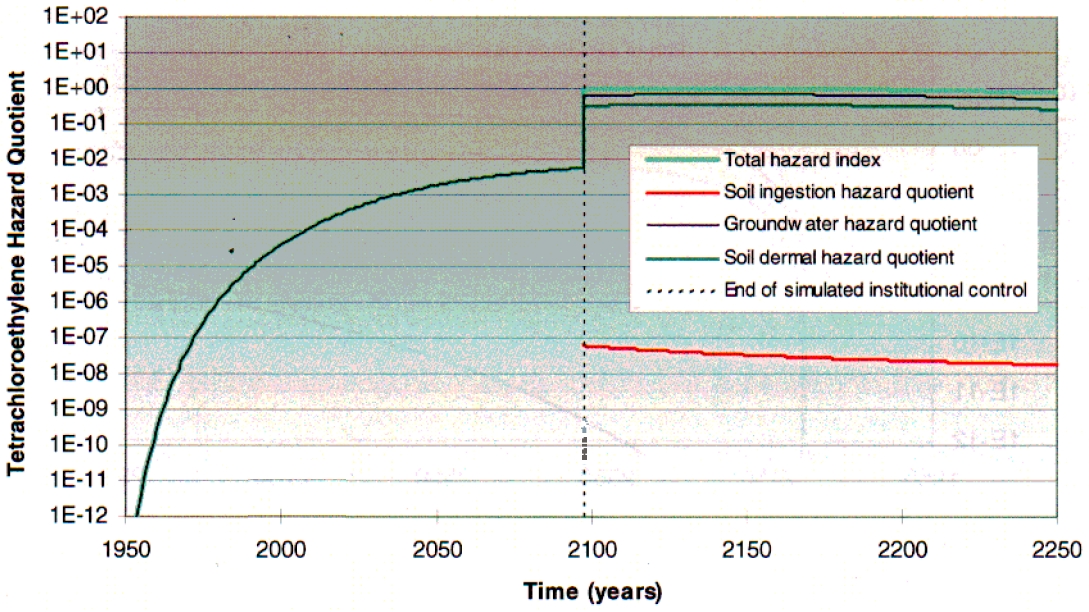

Figure 6-15 Tetrachloroethylene hazard quotient for the hypothetical future residential scenario.

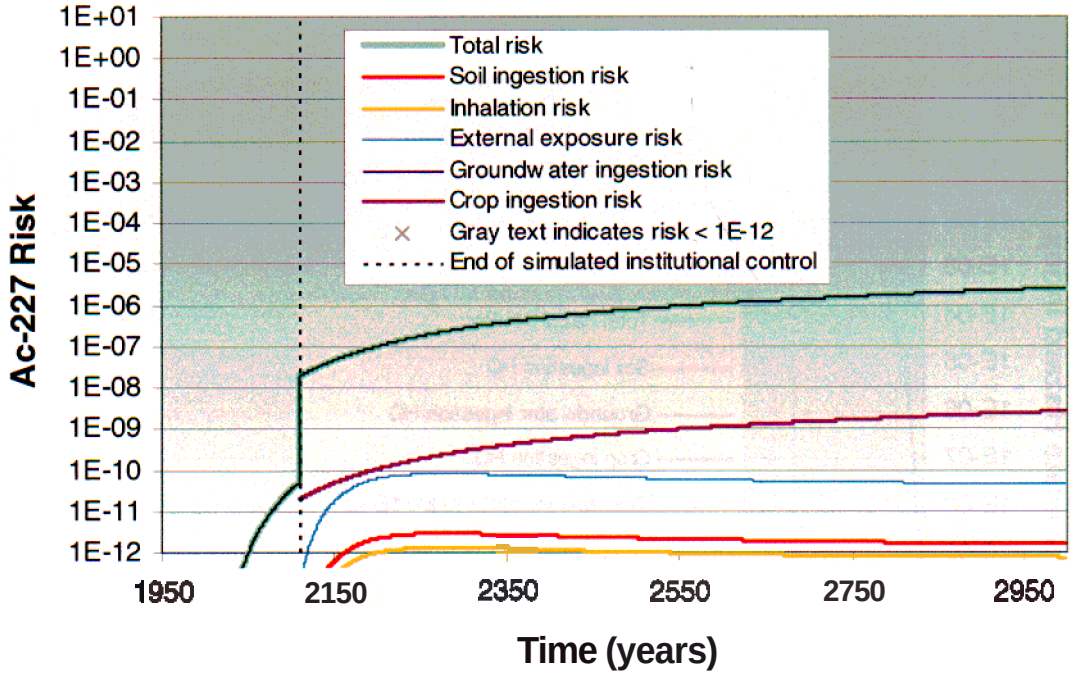

Figure 6-16. Actinium-227 carcinogenic risks for hypothetical future residential exposure pathways.

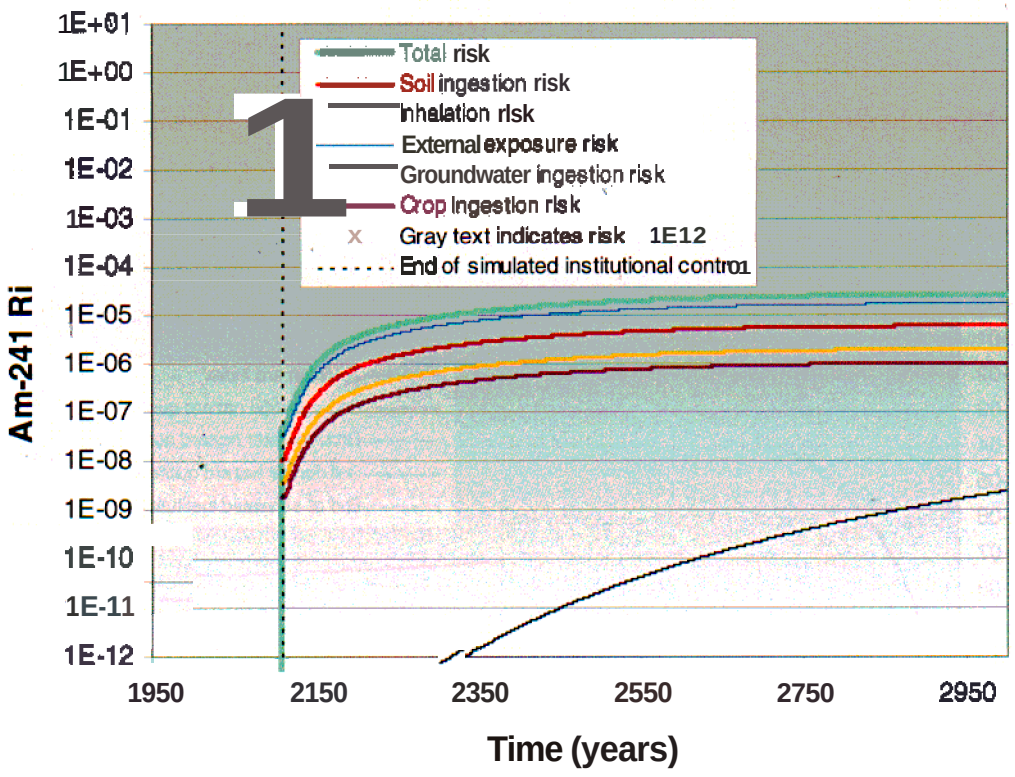

Figure 6-17. Americium-241 carcinogenic risks for hypothetical future residential exposure pathways.

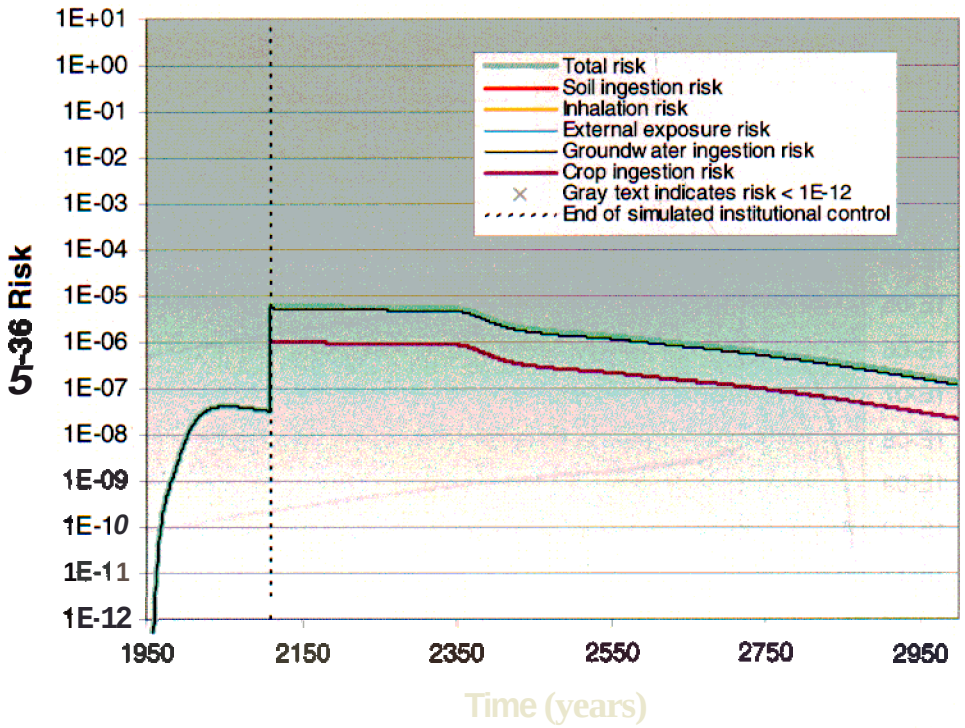

Figure 6-18. Chlorine-36 carcinogenic risks for hypothetical future residential exposure pathways.

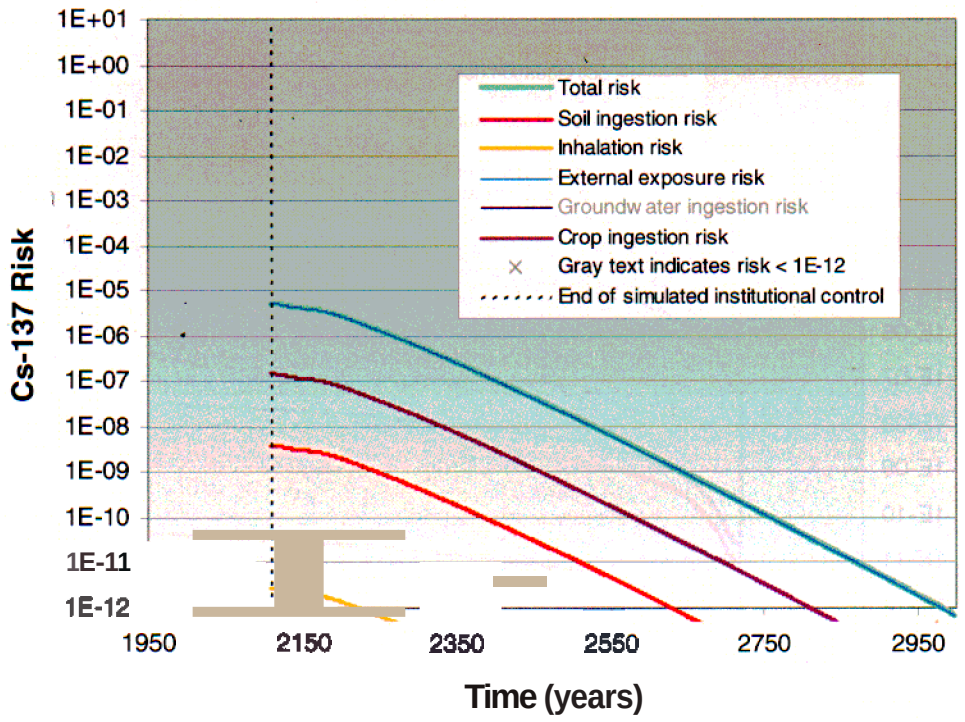

Figure 6-19. Cesium-137 carcinogenic risks for hypothetical future residential exposure pathways.

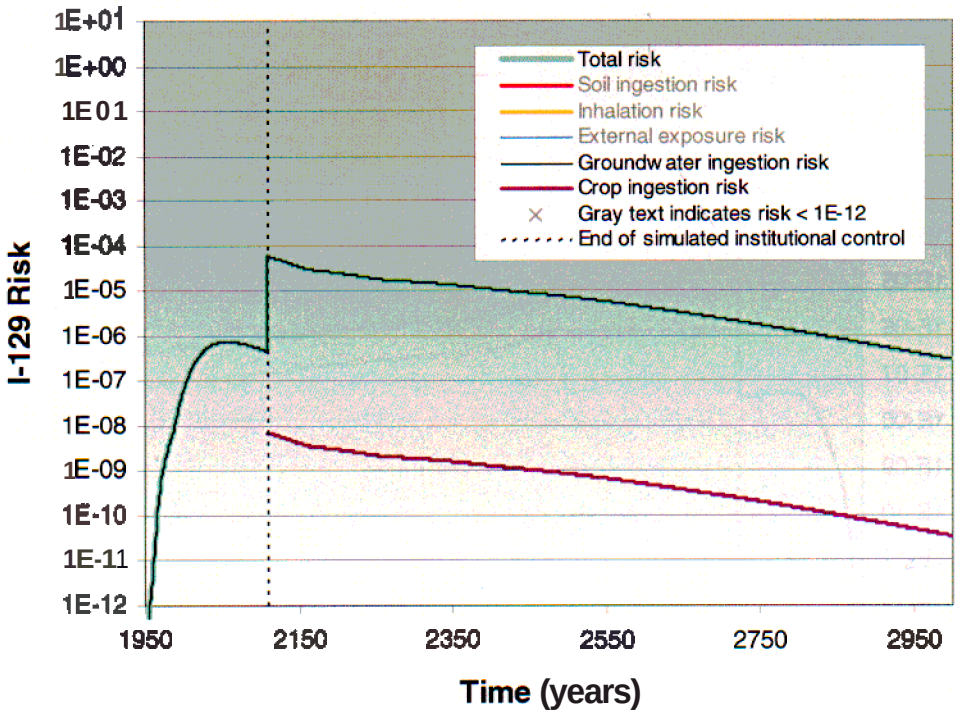

Figure 6-20. Iodine-129 carcinogenic risks for hypothetical future residential exposure pathways.

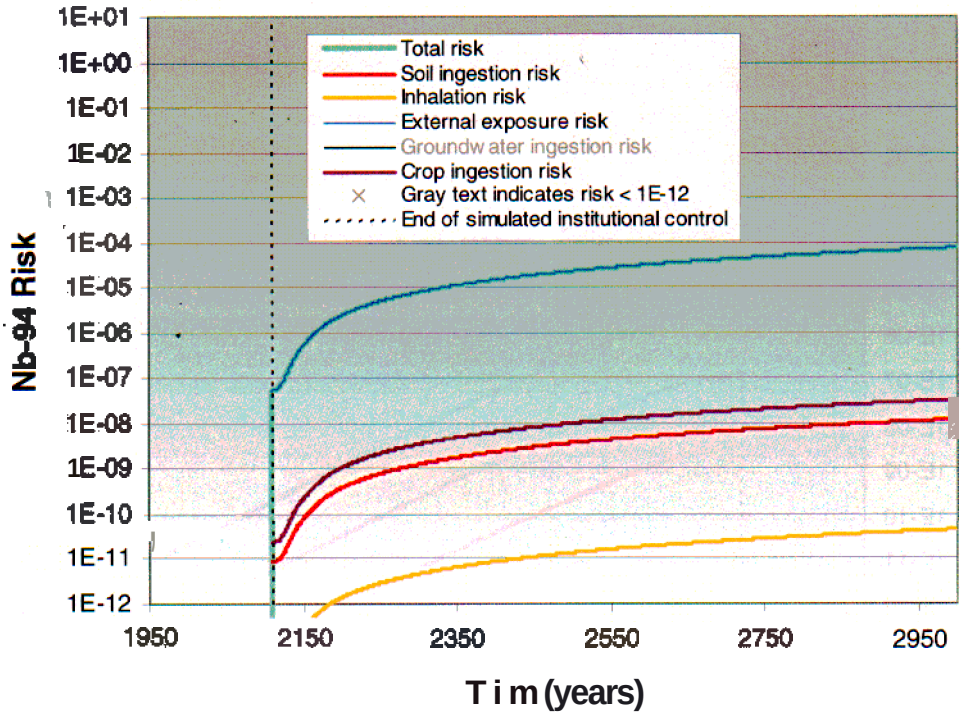

Figure 6-21. Niobium-94 carcinogenic risks for hypothetical future residential exposure pathways.

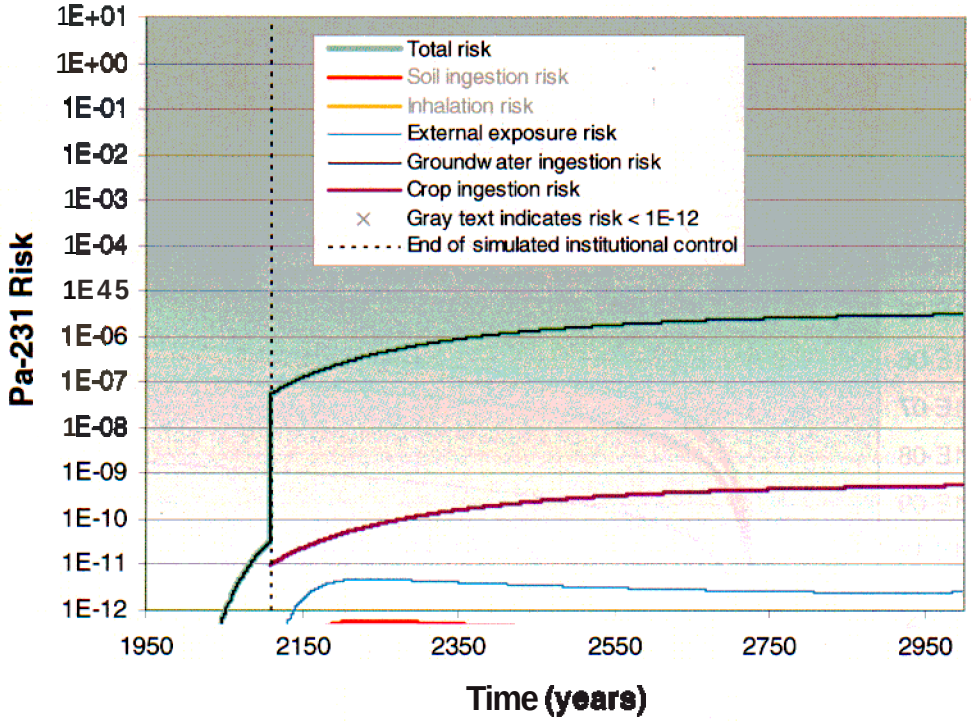

Figure 6-22. Protactinium-231 carcinogenic risks for hypothetical future residential exposure pathways.

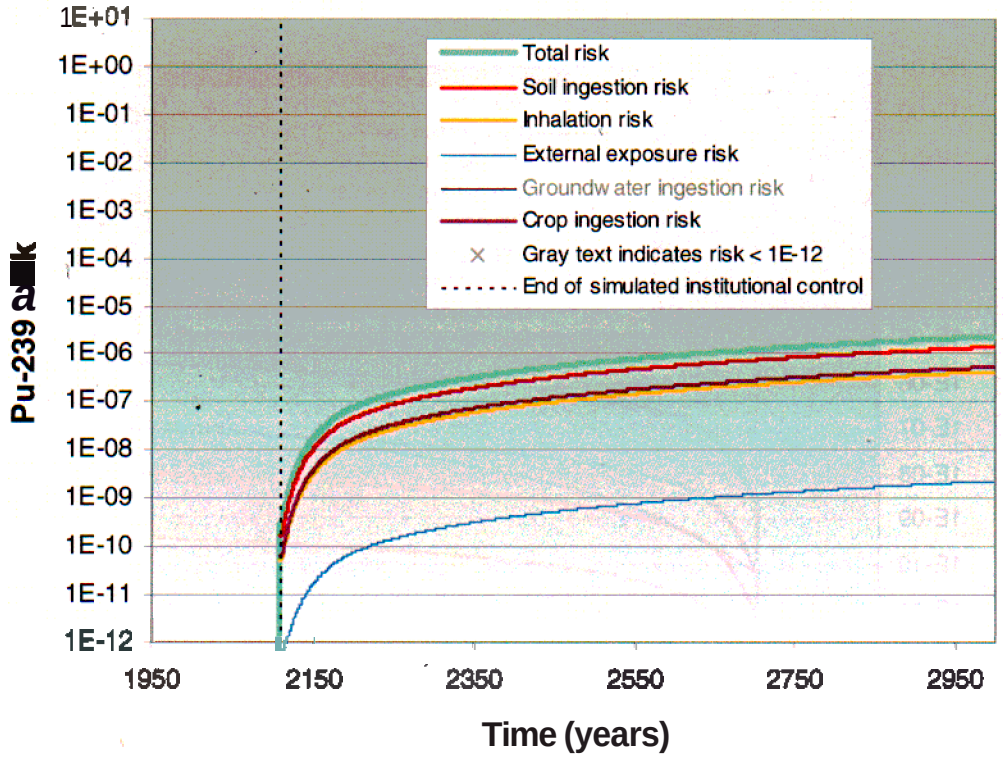

Figure 6-23. Plutonium-239 carcinogenic risks for hypothetical future residential exposure pathways.

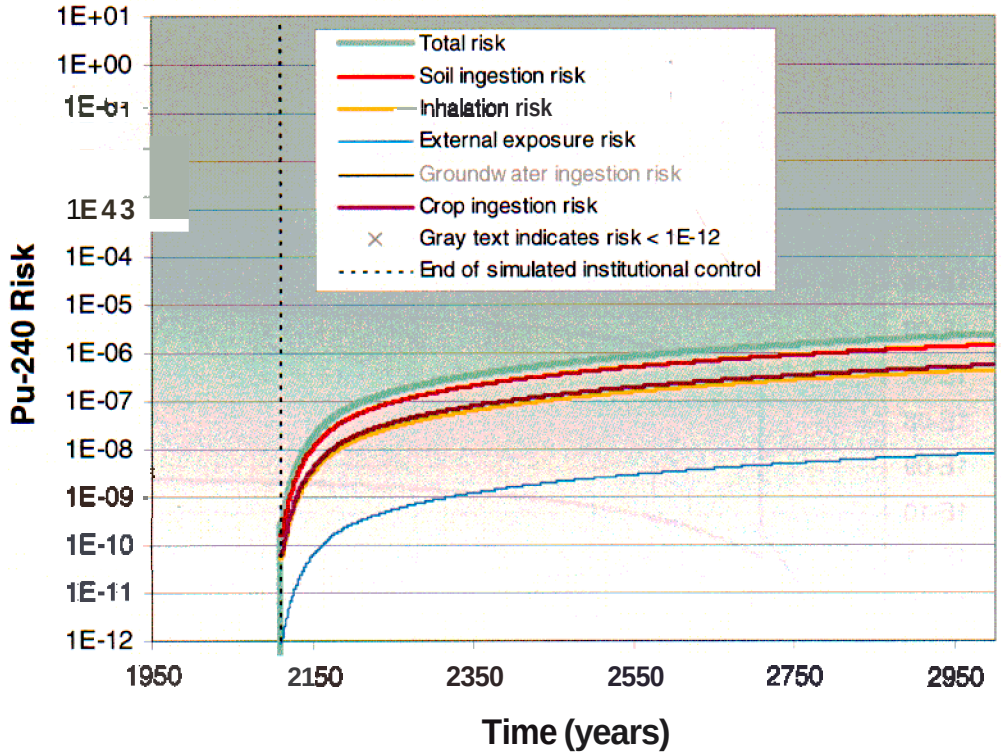

Figure 6-24. Plutonium-240 carcinogenic risks for hypothetical future residential exposure pathways.

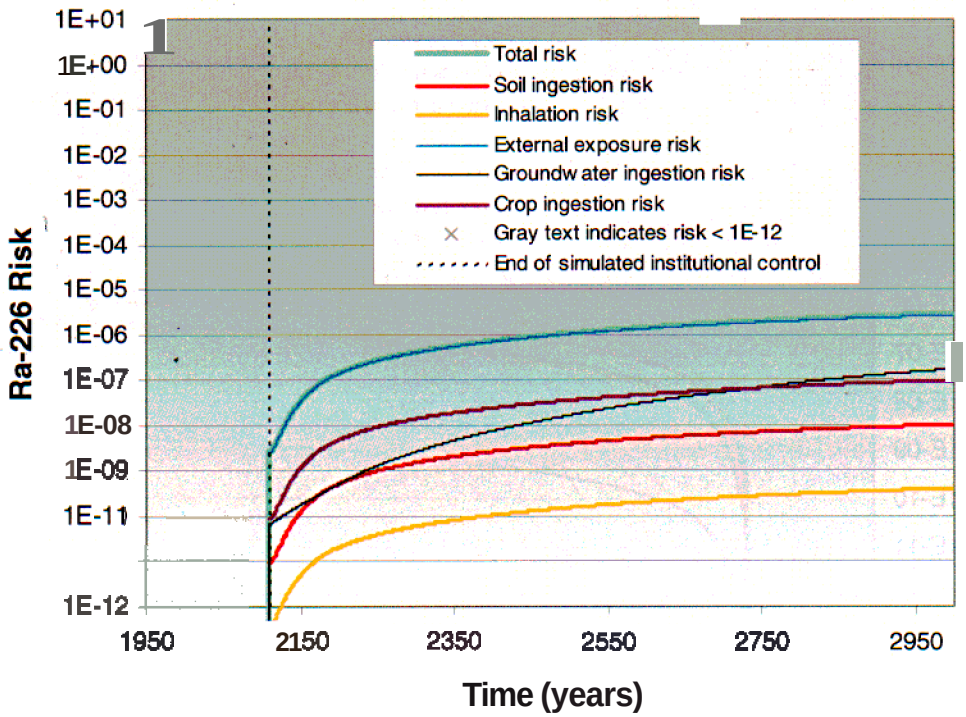

Figure 6-25. Radium-226 carcinogenic risks for hypothetical future residential exposure pathways.

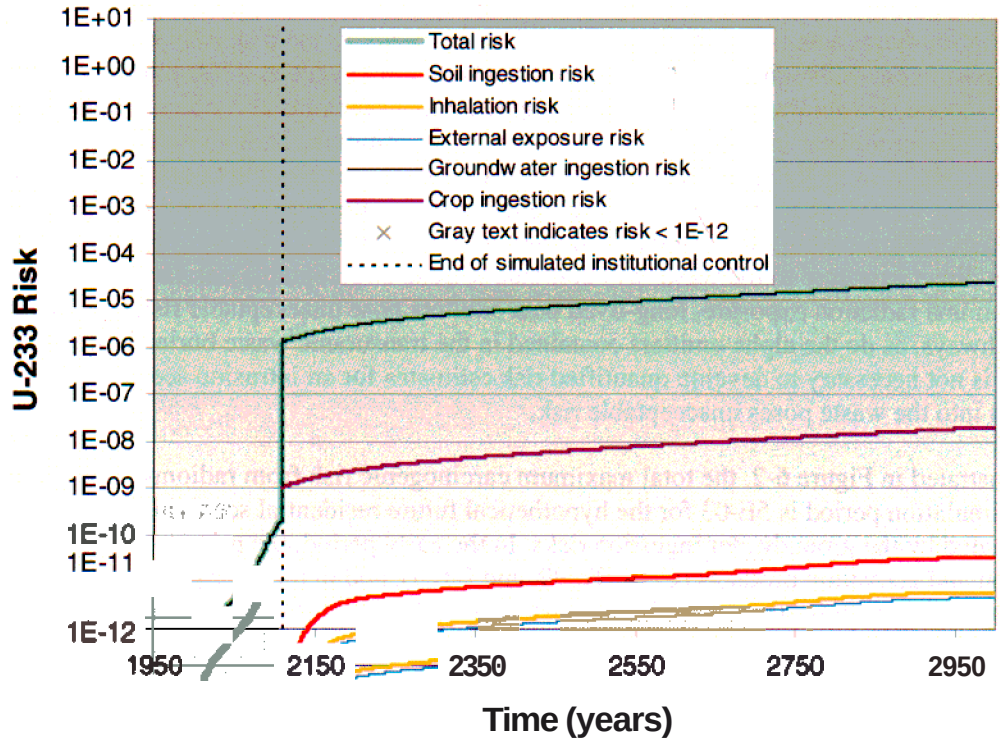

Figure 6-26. Uranium-233 carcinogenic risks for hypothetical future residential exposure pathways.

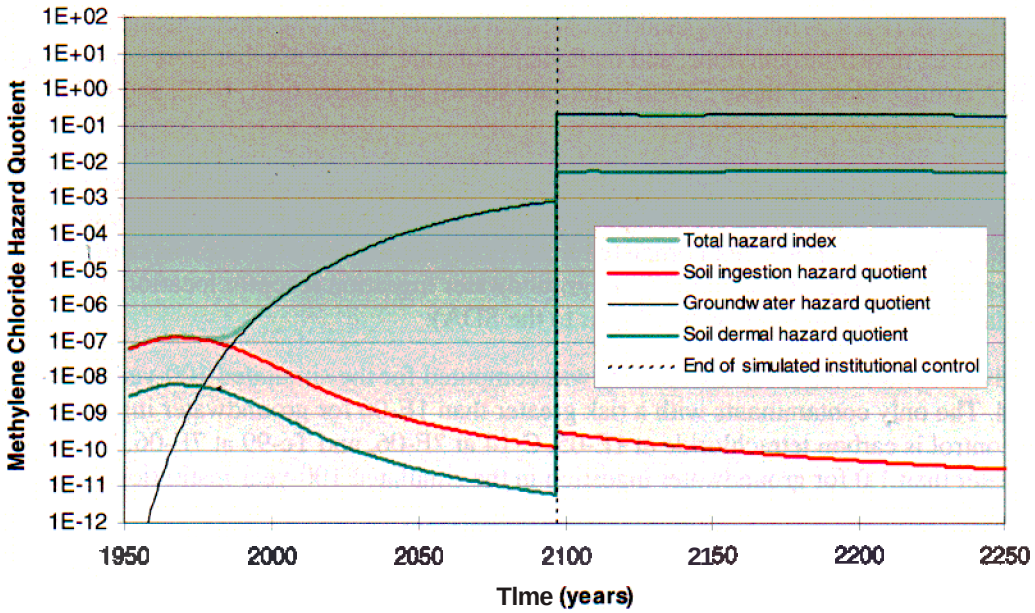

Figure 6-27. Methylene.chloride carcinogenic risks for hypothetical future residential exposure pathways.

Intrusion into the buried waste would produce unacceptable direct exposure risk because of the very high radiation fields associated with much of the waste. Many of the disposals were handled remotely. Exposure rates as high as 1.5E+08 mrem/hr, which would exceed a 100-mrem/year dose rate in less than one second, are shown on historical shipping records. Shipments exceeding 1 rem/hr, which would exceed 100 mrem/year in 6 minutes or less, were not uncommon. Though these high exposure rate waste streams contain short-lived radionuclides such as Co-60, longer-lived beta-emitting radionuclides (e.g., C-14, Ni-59, Nb-94, and Tc-99) also are contained in the waste. Because these isotopes also are gamma-emitters, they generate substantial activity and must be handled remotely. Therefore, intrusion that results in direct exposure to the waste will continue to pose unacceptable risk far into the future. In addition to gamma radiation exposure, long-lived beta emitters pose unacceptable risk from other exposure pathways, as do the alpha-emitters contained in the transuranic waste buried in the SDA. Therefore, it is not necessary to develop quantified risk estimates for an intrusion scenario to determine that intrusion into the waste poses unacceptable risk.

As illustrated in Figure 6-2, the total maximum carcinogenic risk from radionuclides within the 1,000-year simulation period is 5E-03 for the hypothetical future residential scenario. The total estimated risk is dominated by the groundwater ingestion risks. In the early period, the primary risk drivers are mobile fission and activation products (C-14, Tc-99, and I-129), and in the later periods, Np-237, U-234, and U-238 drive the groundwater risks. Risk estimates are still rising at the end of the 1,000-year simulation period for some radionuclides.

The total HI was not computed for the ABRA because contaminants contributing to the HI, nitrates and VOCs, were evaluated with different methodologies. Nitrates were explicitly evaluated in the ABRA while VOCs were evaluated by scaling the IRA results based on inventory ratios (see Section 5.3). Plots of the individual HIs are presented in Figures 6-13 through 6-15.

Similarly, total risk was not computed for chemical carcinogens. The three chemical carcinogens, carbon tetrachloride, tetrachloroethylene, and methylene chloride, are VOCs that were evaluated by scaling the IRA results. Plots of these contaminants are shown in Figures 6-11, 6-12, and 6-27.

Plots of risk and HI show a marked increase at the year 2110 because the exposure scenario changes from occupational to residential. The change in scenario affects exposure parameters (e.g., the exposure frequency is 250 days/year for occupational and 350 days/year for residential), exposure pathways (e.g., crop ingestion was not complete for the occupational scenario but was complete for the residential scenario), and receptor location (e.g., groundwater ingestion receptor location moves from the INEEL boundary to the boundary of WAG 7 next to the SDA).

Groundwater risk at the INEEL boundary was computed for the simulated 100-year institutional control period. The only contaminants with a risk greater than 1E-06 for groundwater ingestion during institutional control is carbon tetrachloride at 1E-05, C-14 at 2E-06, and Tc-99 at 7E-06. No contaminant had an HI greater than 1.0 for groundwater ingestion in the simulated 100-year institutional control period.

Scaled modeling results from the IRA show carbon tetrachloride has a high risk for both inhalation and groundwater ingestion, as shown in Figure 6-11. Dermal contact with contaminated water presents a significant risk in the later periods, as well. The risk from carbon tetrachloride peaks in the year 2107. Because results were scaled from the IRA, the timeframe for the simulated 100-year institutional control period differs slightly. The IRA modeling simulated an institutional control period ending in the year 2097 instead of 2110.

Crop ingestion dominates the Sr-90 risk as shown in Figure 6-5. The risk peaks immediately after the simulated 100-year institutional control period and drops rapidly after that because of radioactive

decay. Soil ingestion produces a risk above 1E-04 during institutional control. Risk from Sr-90 is below 1E-04 by the year 2117, and the risk is below 1E-06 by the year 2336. Groundwater ingestion risks were not computed for Sr-90 because of the short half-life and low mobility.

The greatest risk from C-14 is from the groundwater ingestion pathway as shown in Figure 6-3. The increased release rate and higher mobility used in this analysis, compared to previous results, indicate an earlier peak for C-14. The peak risk is 6E-04 in the year 2278. Comparisons to measured concentrations suggest that the models are overpredicting the release and mobility of C-14 (see Section 5.3 for discussion). C-14 has been detected sporadically at low concentrations in the aquifer.

The U-238 risk is dominated by the groundwater ingestion pathway. As illustrated in Figure 6-10, the risk continues to climb through the 1,000-year simulation period. The risk peaks at 3E-03 near the year 3033 (approximately 1,000 years from the current time), as shown in the plots of the groundwater simulation for a 10,000-year simulation period provided in Section 6.4.3. The U-238 risk is predicted to remain above 1E-04 during the entire 10,000-year simulation period.

The Np-237 risk is dominated by the groundwater ingestion pathway. As seen in Figure 6-4, the risk is still rising at the end of the 1,000-year simulation period. The highest risk during this period is 2E-04. The peak risk is 6E-04, occurring near the year 3913, and is reduced to below 1E-04 by the year 7755 (see Section 6.4.3).

The U-234 risk is dominated by the groundwater pathway. As seen in Figure 6-7, the risk peaks near the end of the 1,000-year simulation period. The U-234 risk peaks at 1E-03 near the year 3193 and falls below 1E-04 by the year 8757 (see Section 6.4.3).

The Tc-99 risk peaks at 4E-04 at the end of the simulated 100-year institutional control period at the year 2110. As seen in Figure 6-6, the risk is posed by groundwater ingestion and crop ingestion (the crops are irrigated with contaminated water). The risk drops below 1E-04 in the year 2422, but remains above 1E-06 during the 1,000-year simulation period. The majority of the Tc-99 is contained in INEEL reactor operations waste. The inventory is still being reviewed (see Section 3.3), and the release rate is uncertain for this waste type. As discussed in Section 5.3, the model greatly overpredicts the measured concentrations of Tc-99 for the current time period. Section 6.6 provides some analysis of the uncertainty in the Tc-99 release rate.

Scaled IRA results for carbon tetrachloride show that the HI is dominated by the groundwater ingestion pathway (see Figure 6-13). The HI peaks at 50 in the year 2105. Because the results were scaled from the IRA, the institutional control period differs slightly from that assumed for the radioactive contaminants.

Scaled IRA results for tetrachloroethylene show that the peak groundwater ingestion risk is 2E-05 (see Figure 6-12). The risk peaks in the year 2097. Because the results were scaled from the IRA, the institutional control period differs slightly from that assumed for the radioactive contaminants.

The nitrate HI peaks at the end of the simulated 100-year institutional control period at a value of 1.0 (see Figure 6-14) because nitrate is rapidly released and is mobile. The nitrate HI is below 1.0 in the year 2120. The HI for nitrate was computed using the infant toxicity values (the most restrictive) with adult exposure parameters.

Scaled IRA results for tetrachloroethylene show a peak HI of 1.0 at the end of the simulated 100-year institutional control period. Scaled results are presented in Figure 6.15.

Scaled IRA results show methylene chloride has a peak risk of 2E-05 from the groundwater ingestion pathway. The risk for methylene chloride is shown in Figure 6-27. Because the results were

scaled from the IRA, the timeframe for the simulated 100-year institutional control period differs slightly. The IRA modeling simulated an institutional control period ending in the year 2097 instead of 2110.

As shown in Figure 6-16, the Ac-227 risk is still growing at the end of the 1,000-year simulation period. The risk from groundwater ingestion peaks at 4E-06 in the year 4073.

Americium-241 has several pathways that contribute significantly to the total risk. The primary pathway is soil ingestion but inhalation of fugitive dust, external exposure, and crop ingestion also contribute to the total risk. As shown in Figure 6-17, the risk peaks near the end of the 1,000-year simulation period.

The Cs-137 risk is dominated by the external exposure pathway. As shown in Figure 6-19, the risk peaks at 5E-06 in the year 2110 immediately after the end of the simulated 100-year institutional control period. Because of the short half-life and low mobility, groundwater ingestion risks were not computed for Cs-137.

The C1-36 risk peaks at 6E-06 and is dominated by the groundwater ingestion and crop ingestion pathways. The C1-36 risk peaks at the end of the simulated 100-year institutional control period in the year 2110, as illustrated in Figure 6-18.

The Ra-226 risk is dominated by the external exposure pathway. As shown in Figure 6-25, the risk is still increasing at the end of the 1,000-year simulation period.

The Pu-239 risk peaks at 2E-06. As shown in Figure 6-23, the primary pathways are soil ingestion and crop ingestion. The risk is still increasing at the end of the 1,000-year simulation period.

The U-235 risk is dominated by the groundwater ingestion pathway. As seen in Figure 6-8, the risk peaks near the end of the 1,000-year simulation period.

The U-236 risk also is dominated by the groundwater ingestion pathway. The risk peaks at 1E-04 at the end of the 1,000-year simulation period (see Figure 6-9).

The Pu-240 risk is dominated by the soil ingestion and crop ingestion pathways. As shown in Figure 6-24, the risk is still increasing at the end of the 1,000-year simulation period.

The Pa-231 risk is dominated by the groundwater pathway. As shown in Figure 6-22, the risk is still increasing at the end of the 1,000-year simulation period.

The U-233 risk is dominated by the groundwater ingestion pathway. As shown in Figure 6-26, the risk does not peak at the end of the 1,000-year simulation period. The risk peaks at 4E-05 near the year 3590. The risk is reduced to 7E-07 by the end of the 10,000-year simulation period (see Section 6.4.3).

The Nb-94 risk is from the external exposure pathway. As shown in Figure 6-21, the risk is increasing at the end of the 1,000-year simulation period.

The I-129 risk is dominated by the groundwater ingestion pathway. As shown in Figure 6-20, the risk peaks at 6E-05 and falls below 1E-06 in the year 2835.

Scaled IRA results for tetrachloroethylene show a peak HI of 1.0 soon after the end of the simulated 100-year institutional control period. Scaled results are presented in Figure 6-15.

6.4.2.1 Maximum Contaminant Levels. Another indication of potential health risks is a comparison of predicted groundwater concentrations to MCLs. The MCLs given in Table 6-7 are taken from 40 CFR 141. The MCL for alpha-emitting nuclides is 15 pCi/L total. The limit has been used for

individual radionuclides as an indication of the potential to exceed the limit. The MCL for beta- and gamma-emitting radionuclides is based on a 4-mrem/year dose. Values used are taken from the 1977 rule as identified in EPA Notice of Data Availability (2000).

Simulated concentrations for carbon tetrachloride, methylene chloride, nitrates, tetrachloroethylene, C-14, I-129, Np-237, Tc-99, U-235, and U-238 exceed the MCLs. Simulated concentrations of C1-36 and Pa-231 are approximately 36 and 10% of the MCL, respectively, and would add to the total beta/gamma or total alpha MCL exceedance.

6.4.2.2 Groundwater Risk Isopleths. Isopleths shown in Figures 6-28 through 6-31 for the total groundwater risk were computed as a function of time by summing the risk from each contaminant in each grid block at the time specified. The isopleths were generated to illustrate risk at the end of the simulated 100-year institutional control period and at the year of the peak risk to show how the risk profile in the aquifer changes with time. Two plots are shown for each period. The local (refined) grid, and the regional grid for the end of the simulated 100-year institutional control period are shown in Figures 6-28 and 6-29. Peak cumulative risk isopleths are shown in Figures 6-30 and 6-31. Effects of the low flow zone south of the SDA and of the additional spreading area water in the vadose zone model that is transmitted to the aquifer, are visible in these plots as the area of reduced concentration immediately southwest of the SDA. As shown in Figure 6-31, the model predicts that the 1E-04 risk does not extend downstream large distances from the SDA.

6.4.2.3 Summary. For all simulated pathways, the highest risk and HI for carbon tetrachloride occur during the first 1,000 years. Risks from uranium isotopes are higher than those from carbon tetrachloride, but occur farther out in time. Risks from Am-241, Co-60, Cs-137, Nb-94, Pu-239, Pu-240, Ra-226, and Sr-90 are from surface exposure pathways. Any method (e.g., a biotic barrier) that inhibits biotic intrusion and contaminant transport to the surface would interrupt the exposure pathway and, thus, eliminate unacceptable risks from those COPCs. Risks from the other radionuclide contaminants, C1-36, Np-237, 1-129, Tc-99, U-233, U-234, U-235, U-236, and U-238, and the nonradionuclide contaminants carbon tetrachloride, nitrates, and methylene chloride are driven primarily by groundwater ingestion. The simulated concentrations for the radionuclides C-14, I-129, Np-237, Tc-99, U-235, and U-238 and nonradionuclides carbon tetrachloride, methylene chloride, nitrates, and tetrachloroethylene exceed MCLs.

All the results presented in this assessment use the models described in Section **5.** Assumptions and limitations of the modeling are discussed in Section 6.1, and the models currently are not calibrated, as noted in the same section. Comparisons to measured data in the vadose zone and aquifer are presented in Section 5.2. An effort to collect data within the waste zone that would allow calibration of the source model is ongoing. Because the source model is the input for the other fate and transport models, calibration of the other models is dependent on calibration of the release model.

Table 6-7. Predicted maximum groundwater concentrations and maximum contaminant levels during 1,000-year and 10,000-year simulation periods.

Contaminant	Peak Concentration for 1,000-Year Period (pCi/L or mg/L)[a]	Year	Peak Concentration for 10,000-Year Period (pCi/L or mg/L)[a]	Year	Maximum Contaminant Level (pCi/L or mg/L)[a]
Radionuclide Contaminants					
Ac-227	2.58E-01	3010	4.35E-01	4073	15[b]
Am-241	1.15E-03	3010	6.60E-02	5355	15[b]
Am-243	7.71E-09	3010	1.30E-03	12010	15[b]
C-14	2.02E+04	2282	2.02E+04	2296	2,000
C1-36	9.74E+01	2110	2.50E+02	1993	700
Cs-137	NA	NA	NA	NA	200
1-129	2.33E+01	2110	8.41E+01	2029	1
Nb-94	6.22E-08	3010	9.97E-01	12010	1,070
Np-237	2.82E+02	3010	4.27E+02	3833	15[b]
Pa-231	8.47E-01	3010	1.28E+00	4153	15[b]
Pb-210	1.96E-02	3010	2.32E-01	11357	Not regulated
Pu-238	1.13E-19	3010	1.47E-19	3273	15[b]
Pu-239	3.20E-12	3010	3.54E-05	12010	15[b]
Pu-240	1.70E-15	3010	2.93E-06	12010	15[b]
Ra-226	2.32E-02	3010	2.33E-01	12010	5
Sr-90	NA	NA	NA	NA	8
Tc-99	5.23E+03	2110	3.87E+04	2007	900
Th-229	3.61E-02	3010	2.45E-01	4354	15[b]
Th-230	3.80E-01	3010	7.52E-01	3833	15[b]
Th-232	1.97E-07	3010	8.27E-03	12010	15[b]
U-233	1.71E+O1	3010	2.89E+01	3593	2.9E+05[c]
U-234	1.06E+03	3010	1.13E+03	3193	1.87E+05[c]
U-235	7.94E+01	3010	7.94E+01	2696	6.49E+01[c]
U-236	7.09E+01	3010	7.19E+01	3113	1.94E+03[c]
U-238	1.62E+03	3010	1.62E+03	3033	1.01E+01[c]
Nonradionuclide Contaminants					
Carbon tetrachloride	1.09E+00	2110	1.09E+00	2110	5.00E-03
Methylene chloride	2.43E-01	2187	2.43E-01	2187	5.00E-03
Nitrates	6.03E+01	2110	6.03E+01	2110	10
Tetrachloroethylene	2.54E-01	2138	2.54E-01	2138	5.00E-03

a. Units are pCi/L for radionuclides and mg/L for nonradionuclides.

b. In accordance with the 15-pCi/L limit on total alpha in 40 CFR 141.15.

c. The uranium limit is 30 μg/L for total uranium.

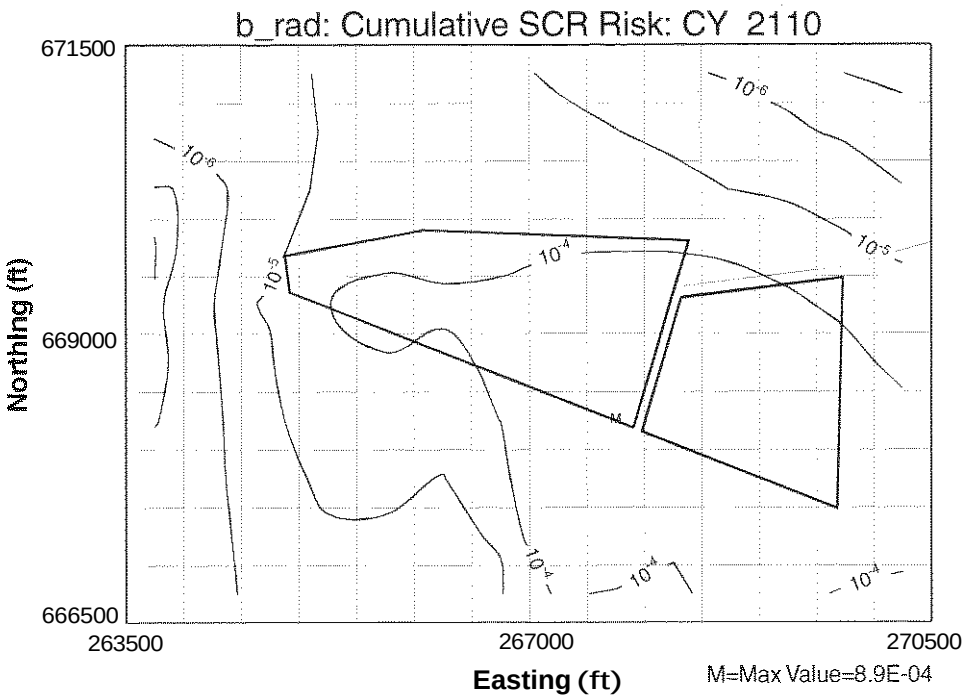

Figure 6-28. Isopleths of cumulative groundwater ingestion risk for radionuclides at the end of the simulated 100-year institutional control period for the refined aquifer grid.

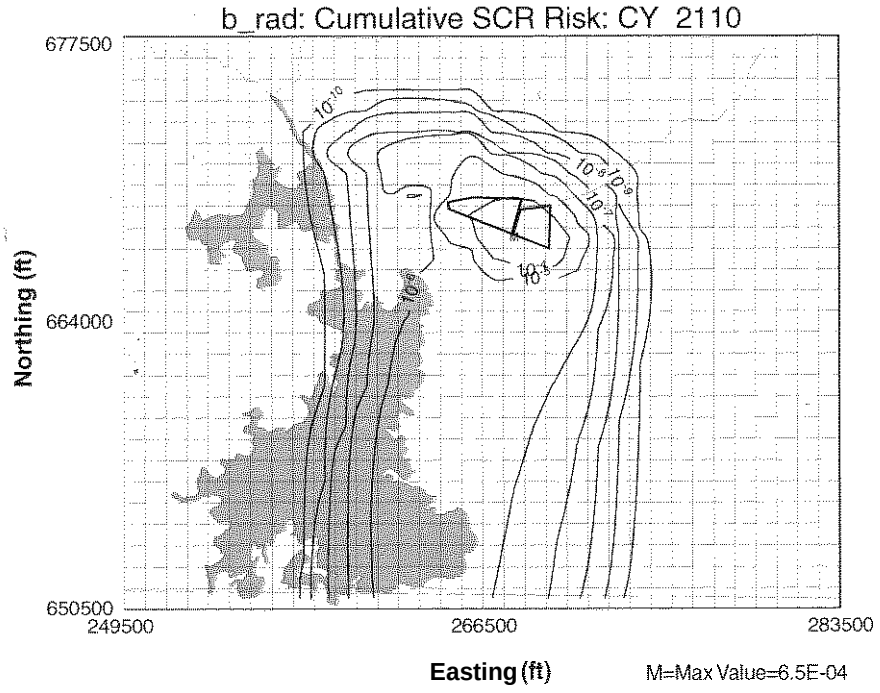

Figure 6-29. Isopleths of cumulative groundwater ingestion risk for radionuclides at the end of the simulated 100-year institutional control period for the regional base aquifer grid.